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THE RELATIONSHIP OF THE INTERVERTEBRAL DISK TO BACK STRAIN AND PERIPHERAL PAIN (SCIATICA)

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DURING the past few years there has been a good deal of writing and much more verbal discussion about ruptured intervertebral disks. One more cause of back pain and sciatica is added to a list which seems much too long. In this paper an attempt will be made to describe most briefly the lesion which has become known as rupture of the intervertebral disk and to discuss certain pertinent points at some length. An understanding of the anatomy, physiology, and pathology of intervertebral disks is a necessary background for this paper. The reader is referred to articles by Keyes and Compere,[1] Joplin,[2] and Beadle[3] for this information.

The syndrome of "sciatica" due to intervertebral disk lesions in the lumbar spine has been described in various papers from the Massachusetts General Hospital[4-7] based on an analysis of 58 proved cases. Love and Camp[8] have reported 38 cases operated upon at the Mayo Clinic in which a ruptured intervertebral disk was found. We know now, as a result of these studies, that intractable low back and sciatic pain is frequently due to the mechanical pressure of intervertebral disk tissue protruding posteriorly into the lumbar spinal canal and impinging on one or more roots of the cauda equina which go to form the lumbosacral plexus.

The symptoms and signs of a ruptured lumbar intervertebral disk are so constant and characteristic that the presumptive diagnosis can often be made before lipiodol roentgenographic studies are made. The typical case is a vigorous man in his thirties, who was perfectly well until, while lifting a heavy weight, he felt something "snap" in his low back and had immediate pain in the lumbosacral region. Sometime thereafter, he

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TABLE I*

Age:	Average 37	Oldest 73 Youngest 16
Sex:	Male 75%	Female 25%
Location of lesion:	Disk between L_{4-5}	50%
	Disk between L_5-S_1	35%
	Disk between L_{3-4}	10%
	L_{1-2} }	5%
	L_{2-3} }	
History of injury:	(a) Immediately preceding disability	50%
	(b) Latent period preceding disability	30%
	Total	80%
Type of injury:	Lifting a heavy weight	50%
	Falls from a height	
	Twisting strain	
	Direct injury by lumbar puncture needle	
Pain:	Constant from onset until operated upon	60%
	Remissions and relapses	40%
Areas to which pain was referred:	(unilateral)	80%
	(bilateral)	20%
	Posterior and lateral thigh	95%–100%
	Posterolateral calf	90%
	Lumbosacral region	70%
	Gluteal and sacroiliac regions	65%
	Lateral border of foot	5%
Signs:	Positive Laségue (limitation of straight leg raising)	100%
	"Sciatic scoliosis"	60%
	Reversal of lumbar curve (kyphos or flat back)	90%
	Local tenderness over the lumbar spinous processes	50%
	Radiating pain on coughing, sneezing or jugular compression	40%
Neurological signs:	Absent or diminished ankle jerk	50%
	Sensory change in the extremity (anesthesia or hypesthesia)	35%
	Motor changes (weakness or paralysis)	15%
	Sphincter disturbance	5%
Lumbar puncture:	Total protein content above 40 mg./100 c.c.	85%
	Total protein content below 40 mg./100 c.c.	15%
	Dynamic evidence of block, either partial or complete	10%
	Cytologic changes in spinal fluid	rare

*This table is compiled from the data available from over 100 case reports of verified ruptured intervertebral disks in the lumbar region. These percentage figures must be accepted as purely tentative estimates until much larger series are available.

developed "sciatica" in one leg, which he describes as a deep-seated pain beginning in the buttock, radiating down the posterior thigh, the posterolateral aspect of the calf, and occasionally going into the lateral border of the foot. Coughing and sneezing, bending forward to tie the shoes, and lying face down in bed cause marked increase in the radiating pain. If an uncomfortable position is maintained for a time, the leg tingles and the foot may "go to sleep." There have been two or three separate periods of disability, the first ones relieved by rest in bed; the last one, however, has yielded to no conservative measures, including back strapping, heat, bed rest, plaster casts, corset, and osteopathic manipulation. On examination the patient is found to stand with his trunk thrust forward and to one side, with most of his weight

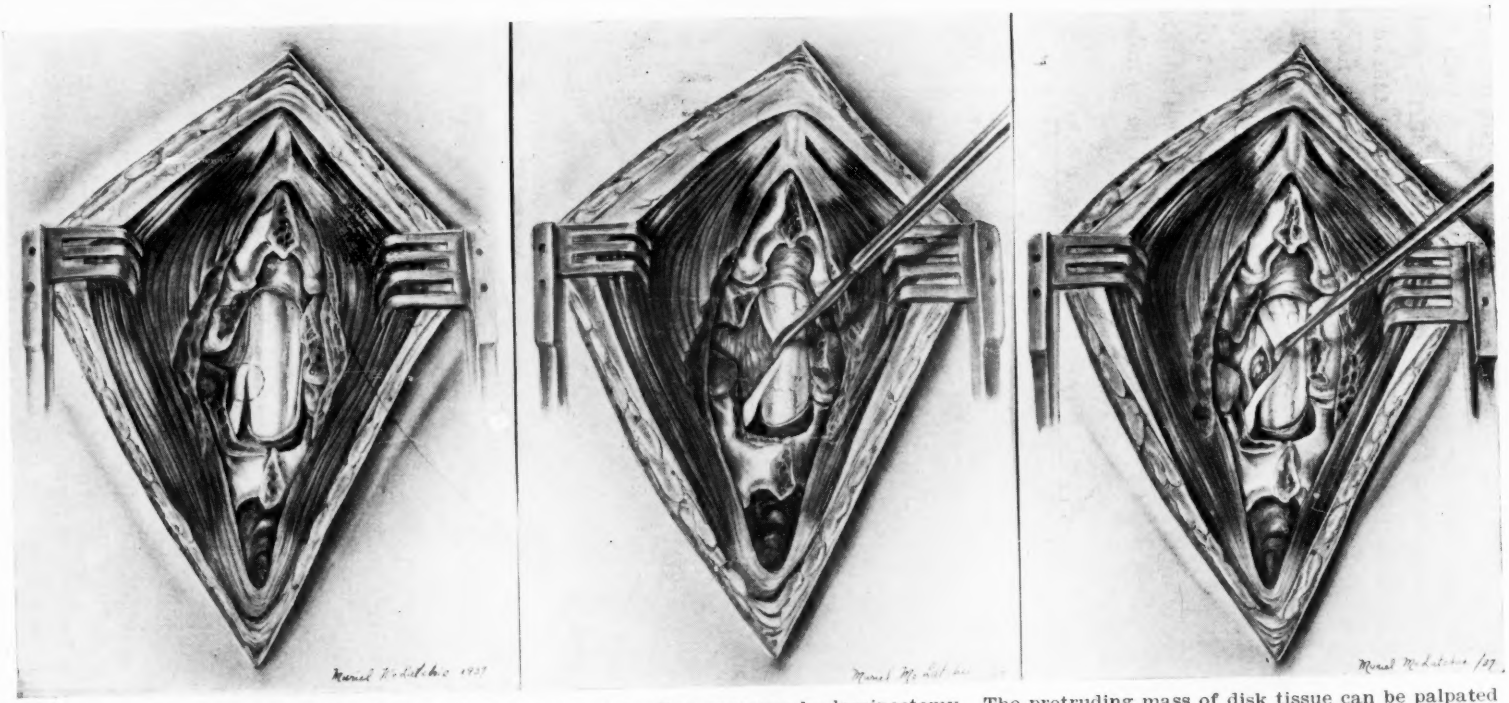

Fig. 1.—*A*, Appearance of the dura and one nerve root after exposure by laminectomy. The protruding mass of disk tissue can be palpated through the dura. *B*, Appearance of the mass after displacing the root and dura to one side. The posterior longitudinal ligament covers the fragment of disk tissue. *C*, The posterior longitudinal ligament has been incised exposing the extruded fragment of disk tissue which can now be removed. This illustration is from the *Annals of Surgery* **106:** 781, 1937, Courtesy of Dr. W. J. Mixter.

borne on the nonpainful leg. The normal lumbar lordosis has been lost and in its place is a fixed reversal of the lumbar curve, with marked prominence of the spinous processes of the third, fourth, and fifth lumbar vertebrae. All motions of the lumbar spine are markedly restricted by fixed involuntary muscle spasm. The patient's finger tips cannot possibly be made to touch the floor if the knees are kept straight. The fixed list of the spine and pelvic tilt are uninfluenced by a lift under either foot. In the sitting position the trunk can be flexed at the hips with ease so that the chest approaches the knees, but observation reveals that the lumbar spine remains in its fixed position and goes forward *en bloc*. The list of the lumbar spine may not be as noticeable, but side bending remains restricted.

Fig. 2.—Case 1. X-ray showing a partially sacralized fifth lumbar vertebra. A woman of 32 years of age who had intractable right sciatic pain of six months' duration, intermittently for two years. She was totally incapacitated, practically bed ridden. Physical examination revealed a fixed list and kyphosis of the lumbar spine; diminished right ankle jerk. Lumbar puncture showed total protein of 33.

The patient gets into bed or onto the examining table with extreme care. Turning over on the table is an ordeal and he is unable to lie face down until a fat pillow is placed beneath the abdomen. In that position on palpation there is found slight but definite local tenderness in the mid line between the fourth and fifth lumbar spinous processes, or at the lumbosacral junction. There is also a little tenderness at the sacrosciatic notch on the affected side. Inspection reveals some atrophy of the buttock, thigh, and calf of the painful side. The ankle jerk is absent on the painful side, normal on the other. The other reflexes are all normal. No definite sensory or motor changes are made out. Straight leg raising can be carried through an arc of 70° on the nonpainful side, and 20° to 30° on the painful side. An attempt to force the leg upward beyond this point causes sharp increase in the sciatic pain. The usual

anteroposterior and lateral roentgenograms of the lumbar spine and pelvis show no changes from the normal, except an abnormally flat lumbar spine (in the lateral view) and possibly slight narrowing of one or both of the lower two intervertebral spaces. A history and physical examination similar to this suggest strongly that the diagnosis is posterior rupture of one of the lower lumbar intervertebral disks.

The next step in diagnosis is the lumbar puncture, which should be done as low as possible, preferably at the lumbosacral interspace. The first 2 c.c. of fluid removed should be sent as a separate specimen for determination of the total protein. After careful manometric tests to rule out dynamic block, further fluid is withdrawn for cytologic study,

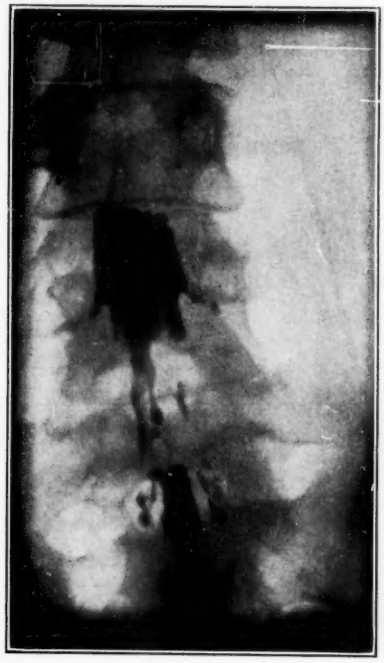

Fig. 3.—Case 1. Lipiodol examination shows almost a complete block at the L4-5 intervertebral disk. At operation June 21, 1937, a large ruptured disk was found at this level and removed transdurally. There was immediate complete relief of symptoms.

Wassermann, and colloidal gold tests. A total protein higher than 35 or 40 mg./100 c.c. is confirmative evidence that the pathologic process is intraspinal. The final step in diagnosis is the accurate localization and visualization of the protruding mass by the use of lipiodol. Using proper technique and a sufficient quantity of lipiodol (4 or 5 c.c.), the roentgenologist should be able to make a preoperative positive diagnosis in over 90 per cent of the cases. Hampton and Robinson's[9] paper should be consulted for details of the roentgen lipiodol examination.

The treatment indicated is laminectomy and removal of the protruding mass of intervertebral disk tissue. The operative technique has been described by Mixter[10] and will not be detailed here. The author

believes that the spine should be fused as the concluding stage of the operation if the patient's condition is satisfactory.

SOME COMMENTS ON VARIOUS ASPECTS OF RUPTURED INTERVERTEBRAL DISKS

Existence and Significance.—Until four or five years ago most orthopedists treated their patients with back disability without suspecting that a ruptured intervertebral disk could be the cause of symptoms. Neurosurgeons had described certain cases of ecchondrosis or chondromas arising from cauda equina, but they were considered to be a rare type of extradural tumor. Careful histologic study reveals that these "tumors" are really displaced intervertebral disk tissue. Their favorite sites of occurrence are the midcervical and lower lumbar spine. The lumbar group is now of greatest interest to the orthopedist, but the author believes that cervical disk lesions account for a certain number of cases of intractable brachial neuritis and stiff necks. No one who has seen these lesions at operation and noted the evidence of root pressure can doubt that they are adequate cause for symptoms. Posterior intervertebral disk ruptures vary greatly in size. The largest ones are capable of producing paraplegia; the smallest ones may cause no symptoms. Table II elucidates the author's conception of the anatomic-symptomatic relationship.

TABLE II

ANATOMIC PATHOLOGY	SYMPTOMS AND SIGNS
1. Small protrusion which causes no nerve root pressure (Schmorl's nodule)	1. No symptoms or possibly low back pain (transient or intermittent) No radiation No neurologic changes
2. Slightly larger lesion causing slight pressure on one nerve root	2. Low back pain Sciatic pain relieved by change in position No neurologic changes
3. Marked pressure on one root	3. Intractable sciatic pain Diminished ankle jerk Subjective numbness and tingling
4. Marked pressure on two or more roots	4. Intractable sciatica Absent ankle jerk Hypesthesia to pin prick (lateral calf and foot) Muscular atrophy
5. Occlusion of the dural sac (partial or complete) with pressure on the whole cauda equina	5. Bilateral sciatica which may disappear if there is irreparable root damage Saddle anesthesia Footdrop Loss of sphincter control

This table is designed to indicate the hypothetical relationship of the size of the ruptured disk to the patient's symptomatology.

The incidence of this entity is still quite unknown. In no way can it be ruled out in any given case except by lipiodol roentgenographic study, done with proper technique. If every patient complaining of low back pain and sciatica were subjected to lipiodol examination, the incidence of this syndrome could be established. However, probably 90 to 95 per cent of the patients who have sciatica recover either spontaneously or after conservative treatment and do not need to be subjected to the highly technical and possibly dangerous lipiodol examination. If adequate conservative treatment fails to relieve a case of "low back strain with sciatica," we have found that an organic lesion is responsible for the continuance of the symptoms in almost every instance and that the organic lesion is often a ruptured intervertebral disk. Lumbosacral or sacroiliac fusion operations done in the presence of intractable sciatic pain will fail to relieve many cases, as the underlying pathologic process is not corrected. Fusions have a therapeutic place in recurring strains in cases which have unstable low spinal and pelvic articulations, but the era is drawing to a close in which one group of orthopedists finds lumbosacral fusion the never failing cure for sciatica, while another school makes sciatica and sacroiliac not only alliterative, but synonymous. Nor should this statement be interpreted to mean that intervertebral disk ruptures and sciatica are synonymous.

DIFFERENTIAL DIAGNOSIS

As noted earlier in this paper, the typical case of ruptured intervertebral disk has a rather characteristic history and physical examination. The difficulty is that the mechanical low back strains frequently have this identical clinical picture. The author has seen cases diagnosed as lumbosacral or sacroiliac strain after careful examination by various orthopedists of the highest repute, cases which were later found to be suffering from pressure of displaced intervertebral disk tissue on one or more roots of the cauda equina.

Almost every author, in his discussion of the differential diagnosis of low back pain and sciatica, stresses the necessity of ruling out cauda equina tumor and details the cardinal reflex, motor and sensory changes which accompany pressure on the cauda equina. A ruptured intervertebral disk may cause sufficient pressure on the cauda equina to produce obvious paralysis, anesthesia, etc., but the majority of these cases have protrusions so located and of such size that only one nerve root is pressed upon and the only symptom is referred pain, all objective neurologic signs being absent. We cannot accept a negative neurologic examination as proof of the absence of this lesion. Neither can we accept the statement that a given case is a "classical picture" of sacroiliac or lumbosacral strain. In either instance the actual pathology may be

intervertebral disk pressure on a nerve root. As Love has said: "There are no physical, neurological, or orthopedic signs which are found alone in cases of protrusion of intervertebral disks. All of the known signs may also be found in other conditions."

There are certain organic lesions of the low back other than tumors which are probably rarer than rupture of an intervertebral disk that need to be considered in the differential diagnosis. Arachnoiditis, characterized by matting together of the roots of the cauda equina, may cause intractable low back and sciatic pain which is usually bilateral. Subarachnoid hemorrhage following trauma may be the cause of this

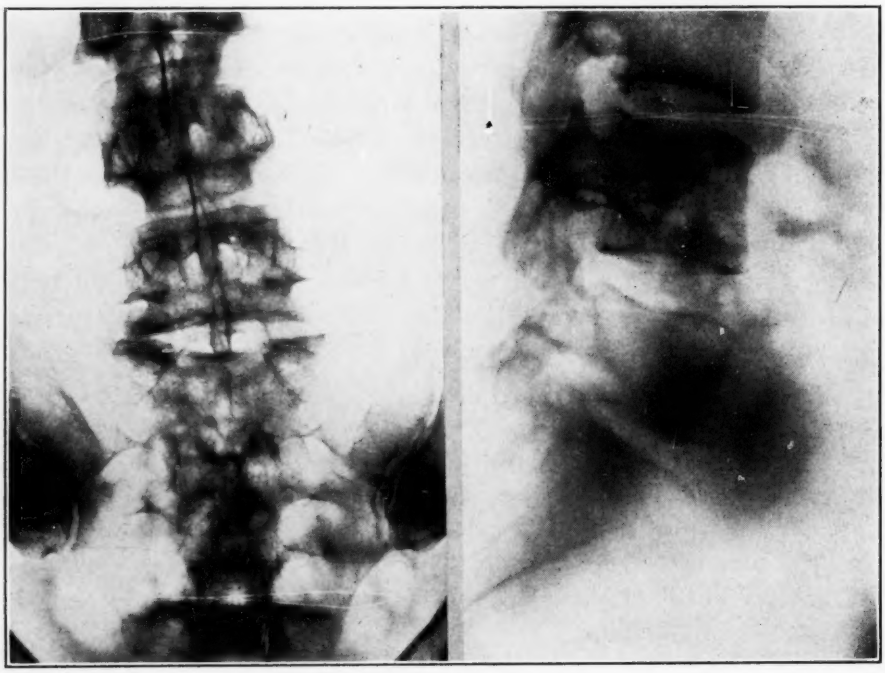

Fig. 4.—Case 2. Anteroposterior and lateral x-rays of a young man, aged 29 years, who had recurrent disabling attacks of low back pain radiating down the right posterior thigh and calf to the ankle. Note the sciatic scoliosis and the reversal of the normal lordotic curve. Lumbar puncture revealed a total protein content of 64 mg./100 c.c.

condition. Lumbar puncture, if done in the area of fibrosis, will show the dynamic alterations of a complete or incomplete block, or it may be impossible to obtain fluid unless the needle is inserted at a higher level. The lipiodol examination is not characteristic, but an incomplete, very irregular, slow filling or a block with an irregular margin is usually noted.

A thickened ligamentum flavum may so constrict the dura as to produce pressure on one or more roots of the cauda equina. Spurling[11] recently has drawn attention to this condition. Can it be due to localized extradural hemorrhage producing fibrosis and thickening of the liga-

ment? Lipiodol examination usually reveals a constricted hour-glass type of filling defect on the posterior and lateral aspects of the spinal canal.

The "facet syndrome" described by Ghormley[12] and narrowing of the lumbosacral intervertebral interspace as emphasized by Williams[13] and Ayers[14] are conditions related in some fashion to the lesion under discussion.

The effect of trauma on the low back is still poorly understood, but our knowledge of the actual pathologic changes which occur is increasing and the future holds promise of further advances.

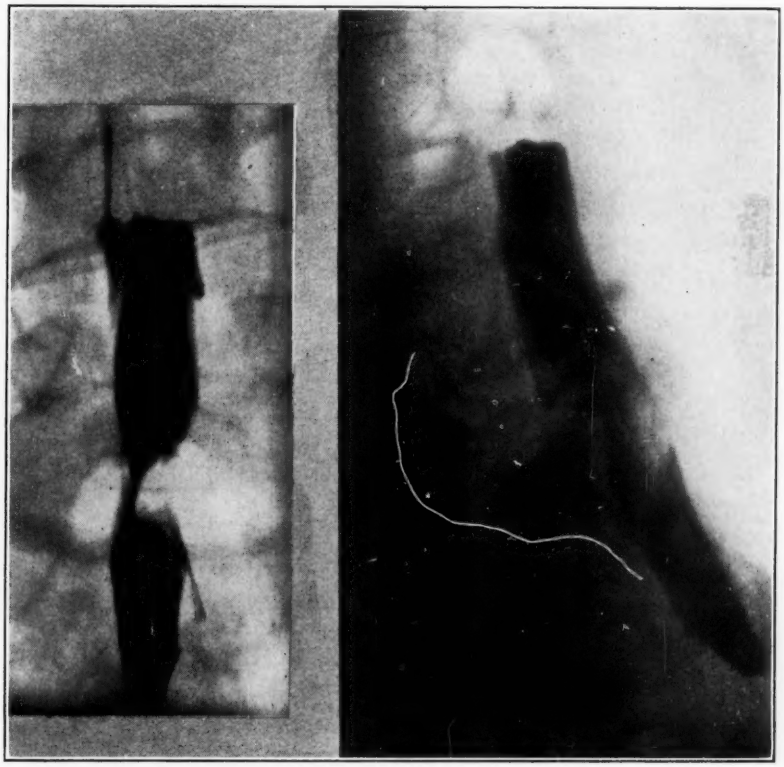

Fig. 5.—Case 2. Anteroposterior and lateral films after the injection of lipiodol. A filling defect characteristic of a ruptured intervertebral disk is demonstrated at the lumbosacral junction. Symptoms were completely relieved by removal of a ruptured fragment 1 cm. in diameter at this level which was pressing on the first and second sacral roots on the right side.

CLINICAL COURSE AND OPERATIVE TREATMENT

The pathologic studies of Schmorl have shown that the normal intervertebral disk mechanism often is disrupted and that extrusion of disk material may and does occur in many patients without causing symptoms. It is only when sufficient disk material is displaced posteriorly into the spinal canal to cause one or more roots of the cauda equina to be pinched that symptoms arise. It is doubtless true that many pa-

tients with this lesion are relieved by conservative measures. The author has under observation one patient with a disk lesion, proved by lipiodol examination, who is perfectly comfortable if the spine is immobilized in a back brace or plaster jacket, but who is incapacitated by severe pain if the support is discarded.

Laminectomy without removal of the protruding mass of disk tissue might relieve symptoms by taking away one limb of the "nut-cracker" which is pinching the nerve root. Spine fusion, if done in a pain-free interval, might prevent recurrence of symptoms and, indeed, has probably been done many times on unrecognized cases of intervertebral disk

Fig. 6.—X-ray of a spine showing what has been described as herniation of the nucleus pulposus, epiphysitis, Schmorl's nodules, etc. *These lesions have no relationship to the posterior ruptures described in this paper.*

lesions. Laminectomy and removal of the ruptured, protruding portion of the disk is certainly the logical treatment for this condition if conservative nonoperative methods fail. The question of whether an attempt should be made to strengthen the spine by fusing the area of laminectomy is still unsettled. If the laminectomy leaves all articular facets intact, the spine is probably little weakened, but, if a facet is removed, I believe that fusion is indicated to prevent symptoms of a weak back from occurring. Two patients in our series of cases have required secondary spine fusion operations for symptoms referable to a weak back after laminectomy. It is technically rather difficult to fuse a spine after laminectomy. We have used a flexible osteoperiosteal

tibial graft to bridge the osseous defect, laying multiple bone chips over the graft. The articular facets are usually destroyed by curettage. Many of our cases have been operated upon by a combined neurosurgical and orthopedic team, the laminectomy and removal of the ruptured disk being done by the neurosurgeon, while the graft is being removed from the tibia by the orthopedic surgeon who then proceeds with the spine fusion and closes the wound. Nursing care is simplified if plaster shells have been prepared preoperatively. After six or eight weeks' recumbency, the patient is allowed up with the back supported by a brace or plaster jacket. Heavy lifting should be prohibited for six months.

END RESULTS

There has been essentially no change in the figures reported in our previous papers. Three out of 58 proved cases are known to be unimproved. There was 1 operative death. The remaining 54 patients are either well or markedly improved. The results reported from the Mayo Clinic and others have been equally satisfactory. The dramatic complete relief which a previously totally disabled patient obtains is most striking.

SUMMARY AND CONCLUSIONS

If a patient is suffering from an intractable sciatica and clinical examination reveals a fixed kyphoscoliosis with limitation of straight leg raising, it is important to consider rupture of one of the low lumbar intervertebral disks in the differential diagnosis. Examination of the cerebrospinal fluid removed by lumbar puncture usually reveals an elevation of the total protein content above 40 mg. per 100 c.c. Lipiodol roentgenographic studies will verify the diagnosis and localize the lesion if it is present with a high degree of accuracy. The operative removal of a ruptured disk is technically difficult, but the results are very gratifying both to the surgeon and the patient.

The organic pathologic changes resulting from trauma to the low back are no longer a sealed book. A ruptured intervertebral disk is not uncommonly the sequel to the trauma of lifting or the ordinary wear and tear of everyday activity. There is some danger that too many patients will be subjected to lipiodol examination or radical exploratory surgery as a result of the untempered enthusiasm of certain men who assume that this entity is the cause of all sciatica. The list of causes of pain referred down the posterior aspect of the thigh and calf is a formidable one. Unless there is evidence of neurologic changes due to nerve root pressure, adequate prolonged conservative treatment, such as is used for any back strain, should be insisted on before lipiodol examination is recommended.

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THE SURGICAL TREATMENT OF LOW BACK PAIN

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MANY cases of pain in the lower back are due to such causes as gynecologic disorders, inflammation of the prostate gland, etc., which do not lie within the scope of orthopedic surgery. Of those patients who have conditions which are intrinsic in the muscles and joints and which are treated by the orthopedist, a small number, approximately 5 per cent, are subjected to operation at the New York Orthopaedic Dispensary and Hospital. Operation is considered advisable in perhaps another 5 per cent, but it is not carried out for various reasons, chief among which is the unwillingness of the patients. It thus is clear that, even in the institution in which spine fusion originated, conservative methods of treatment are used in the great majority of cases. The group for which operation is required is a very important one, however, for it constitutes those patients who have severe, protracted pain, which has not yielded to other measures.

Practically all patients who come to the clinic with this symptom receive a course of therapy consisting of massage, heat, exercises for the correction of posture and for the reconditioning of stiff, painful muscles, and support in the form of belts, corsets, or braces. The large majority are relieved by these measures. Only those who are not cured or improved, who have persistent or recurring pain, and in whom there is some definite defect or disorder in the structure of the lumbosacral region of the spine are advised to have an operation.

It is essential to exercise the greatest care in eliminating other factors, such as gynecologic conditions, prostatic inflammation, spinal cord tumors, and arthritis. This demands the taking of a full history and the making of a thorough physical examination. In all doubtful cases the necessary consultations should be had, particularly with the internist, neurologist, and gynecologist. A spinal puncture with manometric reading and estimation of total protein should be a part of the examination whenever there is any suspicion of a spinal cord tumor. The detection of those cases of arthritis or myositis confined to the lower part of the back often is most difficult and is of the greatest importance in the diagnosis, for the reason that they account for many of our failures.

Frequently the story of pain in other unrelated parts of the body is the most important clue pointing to the presence of arthritis. The x-ray must not be relied upon to exclude arthritis or myositis, because in the stage during which the former is most likely to be confused with

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some mechanical condition, the pathologic changes are not yet demonstrable by this means. Rather widespread tenderness over the muscles of the back and gluteal region, and especially at the site of their origin from the crest of the ilium, is apt to indicate an inflammation due to infection rather than to mechanical strain. The erythrocyte sedimentation rate is a valuable laboratory test which should be used as an aid in making this differential diagnosis. Of course a diligent search should be made for foci of infection, and all obvious ones, such as pyorrhea, apical abscesses, infected tonsils, and paranasal sinuses, should be eliminated. Too often some outstanding fault in the structure of the lumbosacral region is seized upon as the sole cause of pain, without considering that with this there may be a concomitant arthritis or associated condition which may be an equal or more important factor in producing the symptoms.

It is our concept that the pathology underlying painful backs lies far more frequently in the lumbosacral than in the sacroiliac joints. From the standpoint of anatomy and mechanics, there are many reasons for believing that this is true. Man's assumption of the upright posture has placed a strain on the lumbosacral junction, to meet which the joints are but poorly adapted. This point is the junction of the anterior lumbar and the posterior sacral curves and of the mobile with the fixed portions of the spine. The strain, which thus is localized there, is intensified by faulty habits of posture. This alone may be enough to cause pain and is certain to contribute to any other factors which are present. It, therefore, is important in treating these cases to improve the patient's posture as much as possible. It is true, however, that in some cases, particularly those of the markedly slender type, the effort necessary to attain good posture is too great.

Many variations occur in the smaller details of the structure of the lumbosacral joint. It is not asserted that these are the only causes of pain in this region, but it undoubtedly is true that many of them are responsible for an inherent weakness, reducing the competency of the joints to meet the strain imposed upon them, and constituting the important underlying cause of chronic or recurring back pain. Unquestionably some individuals with such structural faults go through life with few or no symptoms. On the other hand, this does not refute the fact that these variations do render this region more susceptible to injury and strain. Although a comparison has not been made in a sufficiently large number of cases to warrant a definite statement, it is quite probable that these variations are found much more often in patients who have chronic low back pain than in individuals who are free from it.

The lumbosacral angle is one of the most important anatomic factors in predisposing to back pain. By this is meant the angle of the plane of the superior surface of the body of the first sacral vertebra with the

horizontal. H. L. von Lackum found in a study of cadavers and x-rays that the average angle is 42°, although the variation from this is great. When the angle is increased, the shearing strain on the ligaments and the intervertebral disk is proportionately greater. The relation of the center of gravity to the lumbosacral joint also is important. This usually is represented by a vertical line passing through the center of the third lumbar vertebra, and, when it lies anterior to the bodies of the fifth lumbar vertebra and sacrum, as happens with exaggeration of the lumbar lordosis, the strain also is increased.

The lateral articulations between the fifth lumbar and sacrum play a very important part in determining the strength and stability of this region. Normally they are concavoconvex and are somewhat intermediate between the coronal plane of the dorsal facets and the sagittal plane of the lumbar. In this respect they vary greatly in different individuals. The more nearly they approach the sagittal plane, the more stable are the joints. When these facets face anteroposteriorly, or in other words are in the coronal plane, the range of motion in the joints is greatly increased and much of their natural stability is lost. This is enhanced by the fact that the facets usually then present a plane rather than a curved surface. The function of these joints not only is to allow a limited range of motion between the fifth lumbar and sacrum but also is to guide the direction of this motion. The inferior articular processes of the fifth lumbar, by hooking over the superior processes of the first sacral, prevent the body of the former from sliding forward on the sacrum. When these articulations are asymmetrical, with one facing anteroposteriorly and the other laterally, as not infrequently happens, the strain is unequally distributed and pain is liable to occur.

Posterior displacement of the fifth lumbar vertebra on the sacrum is a definite entity and frequently is encountered in patients with low back pain. Since the upper surface of the first sacral vertebra slopes forward and the direction of the force upon the fifth lumbar is in this direction, it would seem that a backward displacement could not happen. Willis cited reasons why posterior displacement is not possible and explained the x-rays which apparently showed this phenomenon on the basis of a posterior convexity of the body of the fifth lumbar. He failed to take account of the fact that the fifth lumbar body rests not upon the sacrum but upon the nucleus pulposis, upon which it easily could tip backward if the lateral articulations are so constructed as to make this possible. It is found that in practically all cases of posterior displacement the articulations are of the anteroposterior or asymmetrical type. In order to make this diagnosis, one must have a true lateral x-ray. The amount of displacement may be slight and seldom exceeds one-fourth inch. Its presence in any degree is significant, because in certain positions the extent of displacement may be increased, as can be demonstrated by the comparison of lateral x-rays made with the patient

lying and standing. Many patients with posterior displacement have a history of repeated attacks of low back pain which are apt to come on suddenly, frequently initiated by bending over. They may become increasingly severe and often cause temporary complete incapacity. Pain in the thigh and leg and a so-called sciatic scoliosis are likely to occur. It is believed that the sciatica is caused by direct pressure on the fifth lumbar nerve root in the intervertebral foramen, which is decreased in size when the fifth lumbar moves downward and backward.

Defects in the development of the laminae resulting in failure of ossification are frequent in the fifth lumbar vertebra. The usual sites of these defects are at the midline posteriorly and between the superior and inferior articular processes. The latter are the more important because failure of union then frequently results in spondylolisthesis or forward displacement of the fifth lumbar vertebra on the sacrum. In a large series of cases of spondylolisthesis operated upon at the New York Orthopaedic Hospital such a defect was found in every case. It is possible that such a deformity can result from an upward and forward dislocation of the fifth lumbar articulations, but such an occurrence must be very rare. Defects in the arches of the other lumbar vertebrae are encountered, but they are uncommon. The forward displacement of the vertebral body does not result in all cases of failure of union of the laminae, and numerous instances of this have been seen. In one case x-rays show the presence of the defect without displacement and later films show the subsequent forward slipping of the vertebral body. These defects, even without spondylolisthesis, frequently give rise to pain because of the strain upon the fibrous tissue connecting the two parts of each lamina. It is interesting that in many of these patients symptoms do not appear until late in life.

The process of evolution in the human spine which is tending to produce an upward or cephalad migration of the pelvis accounts for the many numerical variations, most of which are of clinical significance. The usual number of lumbar vertebrae is five, but there are many instances of human spines with four or six lumbar vertebrae. It is apparent that there is a tendency for the fifth lumbar to be incorporated with the sacrum and thus to make contact with the ilium. This explains the occurrence of only four lumbar vertebrae and is an advance in the evolutionary processes. When six lumbar vertebrae are found, the twenty-fifth vertebra in the series has failed to unite with the sacrum and has retained all its lumbar characteristics. It is of mechanical advantage in the upright posture to have a shorter lumbar spine, and thus those individuals with only four lumbar vertebrae have a more stable lumbosacral junction than those having six.

The most important examples of this variation, however, are those in which the sacralization of the last lumbar vertebra is incomplete on one or both sides. The transverse processes are enlarged and make

contact with the lateral mass of the sacrum but form a joint with it instead of uniting by bone. An intervertebral disk is retained between the bodies, and, while the degree of motion between the arches and bodies is small, strains are likely to occur and a painful back often results.

Degeneration of the intervertebral disk between the fifth lumbar vertebra and sacrum is a frequent occurrence. This may result from an injury or may accompany osteoarthritis. Its effect upon the mechanics of the lumbosacral joints is serious. The axis of motion, instead of being through the nucleus pulposis in the center of the disk, is transferred posteriorly to the region of the lateral articulations which no longer function properly. As a consequence, osteoarthritis is liable to develop in them. These patients are subject to sudden attacks of pain in the low back with reflex spasm of the muscles which suggest the possibility of a subluxation. Posterior displacement of the fifth lumbar body also follows in some of these cases and the effect of this and the approximation of the bodies is to diminish the size of the intervertebral foramen to such an extent that pressure may be exerted upon the fifth lumbar nerve root.

Attention has been attracted recently by the reports of Mixter to the consequences of protrusion of the annulus fibrosis or nucleus pulposis into the spinal canal posteriorly or posterolaterally. This may cause pressure upon the cord, cauda equina, or nerve root with resulting pain, motor weakness, changes in reflexes, and areas of altered sensation in the lower extremities. Cases in which definite neurologic changes were present have been very few in the experience of this clinic. If the syndrome is as common as Mixter's cases would indicate, it is believed that a number of them must have been treated by spine fusion alone in our series. Since our results have been good, especially in those patients with a thin intervertebral disk, it would seem that elimination of motion is sufficient in many to relieve the irritation of the cauda equina or nerve roots and that laminectomy and excision of the protruding cartilage may not be necessary. A recent experience in this connection is interesting. An adult male, who complained of low back pain and whose x-rays showed narrow intervertebral disk between the fifth lumbar and first sacral vertebrae, had a high total protein in the spinal fluid and physical signs suggestive of cauda equina pressure. He was subjected to laminectomy and was found to have small varices around the fifth lumbar nerve roots and slight inflammatory changes in the arachnoid. Our interpretation of the findings was that the mechanical strain induced by the derangement at the lumbosacral junction was sufficient to cause these changes without any direct pressure from a protruding cartilage.

It is quite evident that in order to detect the conditions just enumerated a satisfactory x-ray examination of the region is essential. The

technique developed by Dr. Albert Ferguson at the New York Orthopaedic Hospital includes routine anteroposterior, lateral and anteroposterior views taken at an angle of 50° from the vertical. These are supplemented when necessary by other views, such as flexion and extension to demonstrate the degree of mobility at the lumbosacral joint, and lateral views in the standing and lying positions.

Pain does not commonly result from derangements at the lumbosacral junction in persons under 20 years of age. The largest number of patients are in the age group from 30 to 40 years, for it is then that the compensation which was present earlier in life begins to fail and symptoms appear.

The Hibbs' technique of spine fusion has been used in all the cases operated upon at the New York Orthopaedic Hospital. It is our belief that anatomically and physiologically this method is superior, but whatever preference one may have in this matter, it would seem that this technique is better adapted to the lumbosacral joint than the insertion of a bone graft into the spinous processes. Since the spinous processes of the sacral vertebrae usually are small and often very poorly developed, and since in the majority of cases only the fifth lumbar is included in the fusion, the anchorage for a graft often must be inadequate. The desirability of including the lateral articulations and laminae in the fusion is unquestionable. The description of the Hibbs' fusion has been published elsewhere and will not be described in detail. It has undergone certain minor modifications and improvements but it has not been changed materially. Great stress is laid upon the importance of securing a solid fusion of the lateral articulations as well as of the laminae, and to this end the articular cartilage is removed with a chisel; the space left between the processes is filled with a small piece of bone and the articular processes are comminuted with a chisel or gouge. In almost all instances an adequate number of bone bridges can be crossed over between the laminae of the fifth lumbar and sacrum, but whenever the operator feels that an additional supply is needed, it is easily available from the posterior part of the ilium, which can be exposed through the same incision.

In the majority of cases the fifth lumbar vertebra alone is fused to the sacrum. In spondylolisthesis or when some derangement is present in the joints between the fourth and fifth lumbar, the former also must be included. The desirability of retaining the motion in the fourth lumbar joints and the more frequent occurrence of pseudoarthrosis, or failure of fusion, when the fourth lumbar is added have led us to include that vertebra only when there is some definite indication.

Immobilization after operation usually is accomplished by a short Taylor spine brace as modified by Hibbs to secure a better hold on the pelvic region. This is applied immediately after operation and is worn for several months after the patient is allowed up. It offers the ad-

vantages of being adjustable, so that it can be kept snug, and of being removable, thus permitting the patient's back to be bathed daily. X-rays are taken at the end of six weeks after the operation and if the fusion appears to be well advanced the patient is allowed up. In doubtful cases bed rest is prolonged for one or two weeks. Spondylolisthesis cases are kept in bed for twelve weeks because of the necessity for adding the fourth lumbar and because of the greater difficulty in producing a fusion in these cases. Massage and exercises for reconditioning the back muscles are employed with discretion after the period of bed rest. Patients who have had a fifth lumbar fusion should be able to return to a sedentary occupation in from three to four months and to more strenuous types of work in six months.

An end-result study of a large series of these cases has shown that an excellent or good result was obtained in between 80 and 90 per cent. In this group the pain was either completely relieved or was only slight and occasional after the operation, and the patients were able to return to their former occupations. The remainder had only partial or no relief. An analysis of the unsatisfactory group indicates that there were two causes largely responsible for the poor results: first, failure to recognize the presence of arthritis, which naturally was not cured by the operation; and second, failure to obtain fusion. The first cause should be practically eliminated by using every possible precaution to detect the presence of arthritis or myositis. Failure to obtain a solid fusion probably is largely a question of proper technique. There may be a few individuals in whom some of the requisite conditions for making bone are lacking, but in the great majority a properly performed Hibbs' fusion operation will result successfully.

Sciatica in connection with low back pain is a condition worthy of separate consideration. It should be realized that it is a symptom which may be due to a number of different causes and that it is not a distinct clinical entity. It is certain that in some instances it is due to pressure on nerve roots or elements of the cauda equina, and perhaps the most important factor in this group is a spinal cord tumor. This diagnosis must be considered carefully in every case of low back pain with sciatica and must not be dismissed until its possibility has been excluded. When any significant changes in sensation, muscle power, or reflexes are present, a spinal puncture should be a part of the examination. We believe that in many cases of instability of the lumbosacral joint enough pressure is exerted upon the fifth lumbar nerve root to cause sciatic pain. This is particularly true in posterior displacement of the fifth lumbar vertebra. These cases represent another important group of causes of sciatica, and one which is amenable to treatment by spine fusion.

Since Ober described his operation for division of the fascia lata for the relief of certain cases of sciatica, about eighty patients have had this operation at the New York Orthopaedic Hospital. The results have been

reported in detail in another paper. They have been satisfactory in a sufficiently large number to establish this procedure very definitely as one of great value in properly selected cases. In a few pain in the low back was relieved as well as sciatica, but in a larger number this was not the case, and our experience has led us to believe that we should not rely upon this operation to cure low back pain, although it often is a valuable adjunct to whatever treatment is used for the lumbosacral condition, when sciatica also is a part of the symptomatology. Our experience also has caused us to conclude that sacroiliac arthritis and subluxations play a very minor part in the etiology of sciatica as well as in low back pain.

SUMMARY

1. There are many causes of low back pain other than mechanical defects in the lumbosacral joints, important among which is arthritis. Failure to diagnose these conditions accounts for many unsatisfactory results.

2. Conservative methods are satisfactory in about 90 per cent of patients treated by the orthopedist for low back pain. Lumbosacral fusion is indicated in about 10 per cent.

3. The results of spine fusion have been excellent or good in more than 80 per cent of the cases. This proportion can be increased by greater care in excluding arthritis and other conditions not amenable to surgery and by better operative technique.

RELATION OF THE FASCIA LATA TO MECHANICAL DISABILITIES OF THE SPINE

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IT HAS been pointed out in three papers[1-3] that contractures of the fasciae of the thigh bear some relation to low back disabilities and sciatica. In this paper there will be discussed in more detail the mechanical relation of the lower extremities to the trunk and the effect of abnormal contractures of muscles and fasciae of the lower extremities on the spine.

Physiologic Anatomy of the Spine.—The spine is made up of four regions having curves normal to each. The curve of the cervical spine is anterior: the dorsal spine, posterior; the lumbar spine, anterior; and the sacrococcygeal, posterior. The vertebrae are held together and supported by strong ligaments and the different motions and positions of the spine are controlled by muscular action. The muscles are surrounded and held together by strong fasciae and fascial bands. Some of these fascial bands are attached to the bones, ligaments, and muscle aponeuroses. There is an intimate connection between the extremities, both direct and indirect, and the spinal column through fascial and muscle attachments. No very adequate studies have been made regarding the effect of abnormal contractures of these fasciae on spinal movements, deformities, or disabilities.

Contracted Fascia.—It has long been recognized in routine back examinations that limitation of straight leg raising is associated with disabilities of the low back. There may be limitation of straight leg raising in individuals who do not have any discomfort whatever, but these patients often show bad posture, even children. This condition may be present in three or more generations in the same family. In addition to limitation of straight leg raising there is limitation of forward bending of the lumbar and sacral spine, when the knees are held straight, both in the standing and sitting positions. Further examination shows that many of these individuals have abduction contractures[1] and lower leg flexion contractures. In the normal individual lying prone, the heel should be made to touch the buttock; it will not do so when the anterior part of the fascia lata and its intermuscular septa are contracted. If an attempt is made to force the heel on the buttock (the knees must be in contact) when the anterior fascia is contracted, the pelvis will rise from the table and pain down the anterior aspect of the thigh is felt by the patient.

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No explanation has been given to show why a lumbar spine is held in the lordotic position when the posterior structures of the thigh show shortening on straight leg raising. When lumbar lordosis is present, there is shortening of the anterior and lateral soft parts of the thigh; and, since the iliotibial band usually falls in a line from the anterior part of the crest of the ilium to the outer tuberosity of the tibia, this line is in front of the center of the head of the femur so that when the iliotibial band is contracted all the fascia anterior is also contracted. The shortening of the iliotibial band and its anterior fascial connections exerts a pull which must produce flexion of the pelvis on the femur. The sacrum becomes more horizontal than it should, thus increasing the physiologic curves in the spine above.

Most patients with lame backs who have lumbar lordosis associated with fascial contractures are unable to lie in the supine position because the pain is increased in this position. The favorite attitude assumed in bed is lying on either side with the thighs flexed. When there are fascial contractures severe enough to produce lordosis, the dorsal curve is increased and pain may occur along the spinous processes and at the attachments of the interspinous ligaments. This pain is most prominent at the apex of the curve and is often severe and burning in character. It becomes more prominent with increasing age.

Cervical lordosis is usually increased and the vertebral prominens is more prominent, even in young children. Pain in this region is a frequent complaint and is aggravated by certain occupations. It is not the intention of the writer to claim that fascial contractures are wholly responsible for pain over the dorsal and cervical regions because there are several other conditions which may cause the same symptoms. However, when one can rule out these other conditions and no explanation for the subjective symptoms can be found, it is suggested that fascial contractures having an influence on abnormal positions of the pelvis can account for a part, at least, of these patients' symptoms.

In many cases of severe lame backs which have lasted over a long period, the persistent lumbar lordosis may be due to shortening of the interspinous ligaments and contractures of the lumbar fasciae. A release of the contracted thigh fasciae allows the lordosis to disappear. In the mobile lumbar spine the excessive lordosis disappears rapidly and usually without symptoms. In the "fixed" lordotic spine the curve disappears slowly and is attended by symptoms of low back strain. This must be expected since the mechanical stretching of the lumbar fascia and interspinous ligaments must produce irritation at their bony attachments.

It has been stated by some that everybody has an abduction contracture and by others that no one has such contractures. The same could be said of limitation of straight leg raising. The straight leg raising sign has been used so many years that no one who knows any-

thing at all about lame backs ever omits to perform the test. However, there have been instances when this sign has not been found to be increased because the examiner did not check his test against the flexion roll of the pelvis.

History.—When a patient presents himself, complaining of trouble in his back, it is necessary to secure a complete history of his complaint, duration and number of attacks, and duration of preceding attacks, if any. The onset and its character are important. It is necessary to know about pain, its localization, and its character and severity; whether the pain is in the back or whether it radiates or is referred; what increases or diminishes it and what is the effect of certain positions on the induction or relief of the pain. It is well known that sneezing may precipitate a low back pain and sciatic pain which may be exquisite and out of all proportion to the apparent cause. Pain along the sciatic nerve may be so severe that the patient is extremely uncomfortable in any position.

The history taking must not be limited to the local condition but must be general and thorough since pain in the back is so often due to abnormal conditions elsewhere in the body. One sometimes sees patients with kidney and bladder lesions (especially calculi) who have had their backs strapped and supported for a long time. A good history and physical examination would have helped to make a proper diagnosis. By the same token it should not be assumed that all cases of sciatic pain are due to tight iliotibial bands and contracted fasciae. There is no surer way to bring discredit on a new procedure than to use it as a waste-basket by making snap diagnoses followed by surgery which gives no relief. Always bear in mind that there are many lesions giving rise to pain down the leg; e.g., malignant disease of the spine and pelvis, Brodie's abscess of the femur, or neuromas of the nerve.

Examination of the Back.—First note from the back, side, and front how the patient stands. Note abnormal exaggerations of all the physiologic curves of the whole spine, whether there is a list of the body laterally and whether it has any relation to sciatic pain. Note whether it is possible to change the list to the opposite side. (Sometimes the list is alternating and can be changed by the patient.) Is the list increased on forward bending of the spine? Note whether there is lateral motion toward or away from the listed side and, if any, where it occurs; i.e., above the list, in the area of the list, or below it. Is there restriction of forward bending in the spine and is it due to muscular and fascial shortening or to muscle spasm or ankylosis? The spine should be examined for limitation of motion in all directions, both in the erect position and in the sitting position with the knees flexed at a right angle. Limitation of motion is very marked in forward bending of the spine when the lower legs are extended if there are contractures of the posterior and lateral thigh structures. This forward motion in the same position is restricted when there is a low back lesion protected by muscle spasm.

When all the spinal motions have been tested in all positions then the tests as described should be done; i.e., straight leg raising, abduction, and knee flexion with the patient prone. The relation of limitation of straight leg raising, abduction, and lower leg flexion contraction to the mechanics of the pelvis and spine has never been worked out satisfactorily. Probably most physicians have been led to believe that Laségue's sign is a result of a low back disability. Since this sign is often present in both legs, without associated symptoms and signs in the back, it would appear that it might be possible that patients having limitation of straight leg raising (which is more limited when there is a low back lesion) belong in a class more subject to such disabilities, especially when there is added to this the abduction and leg flexion contractures which, as explained before, are caused by contracted fasciae.

If one asks why this should be so, it can be answered in several ways: (1) a patient who cannot flex his lumbar spine and pelvis to an angle more acute than a right angle while sitting with his legs straight, or (2) one who cannot reach the floor with his hands in forward bending, when his knees are straight, is putting his low back under a more severe strain than the one who *can* flex his trunk and pelvis until his head touches his extended knees. In other words, the patient with short fascial structures in his leg-pelvic junction puts them in more severe tension in these positions than the person does who has a perfectly normal forward bending.

As a result of sudden overstretching or bad positions, the "give" is going to occur at the point less likely to stand the greatest pressure and that place is the low back. If there happens to be an inequality on the two sides in the contractures or if there is more on one side, then other forces are produced in this region so that there may be produced a train of symptoms in one side only. Can anyone explain why a sneeze or a slight twist of the body may produce an extremely lame back with the severest pain along the sciatic nerve when the x-ray will show a perfectly normal spine? Conversely, why does not every back showing marked lesions by the x-ray present such severe symptoms?

Patients who have low back symptoms due to fascial contractures are usually tender to pressure over the posterior trochanteric region, and sometimes the tenderness amounts to real pain. There is often tenderness also over that part of the iliotibial band between the ilium and the trochanter, and, in many cases of sciatic pain, pressure over the band in this region will often increase the pain along the sciatic nerve. Similar observations on referred pain in low back disabilities have been made.[4] In all these cases there is spasm of the upper bundles of the gluteus maximus muscle which can be felt very readily by the examiner. Also there is often soreness or tenderness on pressure in this part of the muscle. It is quite possible that these symptoms may account for some of the limitation of straight leg raising.

X-ray Examination.—X-ray examination should include an anterior stereoscopic view of the whole pelvis and the lumbar spine, a true lateral view which should include the lumbar spine and the *whole* sacrum, oblique views to show all the articular facets of the lumbar spine, and a lateral standing view of the lumbar and sacral regions in order to show changes in the relation of the lumbosacral angle.

Examination of the Lower Extremities.—Limitation of straight leg raising is seen in sciatica which is unilateral, but who has not noted limitation of straight leg raising in many instances on the contralateral side where no sciatica exists? There are also many patients who have marked limitation of straight leg raising on both sides who have never had a lame back. When these patients do have lame backs, the limitation of straight leg raising is more pronounced and it decreases as the back improves. However, straight leg raising in most of these cases is not normal at any time. Limitation of straight leg raising may be due to hereditary factors, irritation of the hamstrings, pain in the sciatic nerve, or contracture of the posterior part of the fascia lata and its posterolateral intermuscular septa, or to spasm of the gluteus maximus muscle.

Ely's sign was originally supposed to mean that, when the knee was flexed with the patient lying in the prone position, the pelvis would rise from the table if there was low back or sacroiliac irritation. The knee-flexion, prone position sign is often present in patients who have no signs of any low back lesions. The pelvis rises in these cases of limited knee flexion because the *anterior structures of the thigh are contracted.* In such cases when passive knee flexion is attempted, the pull of the anterior structures, which are attached to the anterior part of the crest of the ilium, anterior superior and inferior spines, flexes the pelvis on the femur. If the examiner places the fingers of his free hand on the soft parts of the thigh, just below the anterior spine, a bowstring tightening can very easily be felt. In addition to this the patient complains of pain when the knee flexion is forced. The cause of this contracture is probably hereditary or due to adaptive shortening which might possibly occur in utero or from bad positions of the young infant's legs before the walking age. It must be remembered that the infant sleeps with his thighs flexed and when he creeps the thighs are flexed.

The method of eliciting abduction contracture has been given in detail in a previous publication.[3] When abduction contracture occurs, the iliotibial band can be easily felt and in the severe contractures it will be seen standing out on the whole lateral aspect of the thigh. If one attempts to flex the knee beyond a right angle while the thigh is in the abducted position, *the abduction increases* when the anterior part of the fascia is tight. One can imagine that the fascia lata is a snug sleeve enclosing the thigh muscles and that the iliotibial band represents the seam in the sleeve. The fascia lata not only surrounds the muscles

of the thigh as a whole but also has compartments surrounding the individual muscles and also many intermuscular septa which extend down deeply between muscles and which are attached to the surfaces of these muscles and to the femur. For example, it surrounds the belly of the tensor fasciae latae and there are extensive trabeculations of fascia dipping down between the tensor fasciae latae and sartorius, between the tensor and the gluteus medius and between the gluteus medius and gluteus maximus. These trabeculations of fascia are very thin and strong between these muscles and over the posterior aspect of the gluteus maximus.

The tensor fasciae latae does what its name implies. The iliotibial band in its lower two-thirds is also the tendon of the tensor. Three-fourths of the gluteus maximus tendon is inserted into the iliotibial band below the trochanter and about opposite the lower end of the tensor. The upper anterior third of the gluteus maximus is either contracted or in spasm when abduction contracture is present. In cases of abduction contracture the iliotibial band can be easily palpated just below the crest of the ilium when the patient is lying on his back. Sometimes the band in this region feels like a narrow bone to the palpating fingers. If the band is tight, another way of testing the tightness is to flex the thigh on the opposite side and then adduct the extended leg. In such instances the pelvis will begin to tilt laterally with the adduction, since the iliotibial band from its attachments to the leg and pelvis acts as a stay or guy rope when the leg and thigh are extended.

It has been pointed out in previous papers[1-3] that lumbar lordosis is usually increased if the iliotibial band and anterior fascia are contracted and that flattening of the back occurs if the iliotibial band is behind the trochanter and contracted. Lame backs and sciatic symptoms may be present in either case. It has long been recognized that other conditions producing lordosis, such as the so-called pregnancy back or the low prominent abdomen, are frequently attended by low back pain. If this is so, then it seems reasonable to suppose that any other factor or factors which increase the lordosis may also produce low back symptoms. The converse is also true; i.e., when the lumbosacral angle is more flat than normal or the lumbar curve is reversed, low back symptoms occur.

Diagnosis.—There are many other conditions in which backache and radiating pain may occur. These have been referred to in other articles by numerous authors before and in this number of SURGERY, therefore these conditions will not be discussed here since it would involve unnecessary repetition. It should not be supposed for a moment that abnormal contractures of fascial bands account for all low back disabilities, any more than a ruptured nucleus pulposus,[5] a sacroiliac lesion, or a contracted pyriformis muscle[6] is responsible for all sciatic pains.

To go a step further, it must be assumed that there may be factors, at present not recognized or understood, which may be responsible for some of the lame backs which occur. The unknown or unsolved problems become fewer and fewer as new symptoms and signs are found and discussed. The dangers of adding new information, however, are great because there is a tendency, on the part of some of us at least, to rush hastily into the new field with the idea that the new or the latest is the final word or solution, without taking into consideration that there are many other things which must be ruled out, at least before considering the new. It must always be borne in mind that there is no single factor which will account for all backaches. When physicians jump to such conclusions and make snap diagnoses, sooner or later, they are bound to get into trouble which may be very serious to a patient and at the same time may bring unjust discredit upon some procedure which has only limited possibilities at best and is only the answer to one part of the whole problem. In other words there is no one "cure all" for multiple difficulties, even when these are confined to a definite region.

Treatment.—The treatment of mechanical difficulties of the back, which are associated with fascia lata contractures, depends, first, on removing or minimizing the contracture and, second, on securing a return to the normal physiologic position of the spine. It ought to be obvious to any physician that no amount of postural exercise will correct a mechanical difficulty if there is any mechanical obstruction to such correction. When the obstruction is removed, exercises should be begun. The exercise dosage depends on the general musculature of the patient, the degree of discomfort present, and the strength of the muscles which maintain the spine in its correct position.

There are several methods of approach in the management of lame backs of the type discussed in this paper: (1) rest in bed, (2) rest plus local immobilization, (3) traction, plus rest and immobilization, (4) fixation in a spica, (5) physical therapy to relieve the fascial contractures, and (6) operation on the contracted fascia lata.

1. *Rest.*—Rest in bed is imperative in cases of severe pain in the sciatic nerve. The bed must be firm, not the so-called comfortable mattress, and it should not be too hard, since a bed that is too firm is uncomfortable when the patient lies on his back.

2. *Rest Plus Local Immobilization.*—It is well known that in some instances adhesive plaster strapping, effectively applied to the low back, gives relief from back symptoms and sciatic pain. Such relief is not due to the immobilization of the spine or the sacroiliac joints because it is not effective enough to accomplish such a result. However, it does pull the buttocks closer together and in this way probably relieves the spasm in the upper third of the gluteals which is so often present in these cases. A well-constructed corset which fits in a proper manner will also

relieve a great many of these patients. A belt designed to relieve because it can be tightened up is not of much use and does not help very often. Belts covered with extra gadgets are even more useless as they are usually ill fitting. The only way to have a proper fitting corset is to teach the corset maker how to cut his cloth so that the corset holds and supports the abdomen and back. The secret of this is in letting the crests of the ilia out into the cloth. Any corset or belt that compresses the iliac crests will never give adequate support for a lame back.

3. *Traction.*—Traction probably stretches out the spasm in the gluteus maximus muscle and the hamstrings.

4. *Fixation in a Spica.*—A plaster spica puts the lower extremity at rest and allows the spasm in the gluteus maximus muscle and hamstrings to subside.

5. *Physical Therapy.*—Patients who have bad posture or who have pain in the low back due to fascial and muscular contractures should have exercises[1] prescribed which are designed to stretch those shortened structures as follows:

A. The patient stands with his affected side toward a table or some other convenient object, which is about 3 feet in height, so placed that it does not slide.

B. He should be at least 2½ or 3 feet away from the support. He then grasps the edge of the table with his hand.

C. He should then bend his hip laterally toward the table, being careful not to rotate or twist his body or hips, until the body and legs form an arc of a circle. This position must be held for six or eight seconds. The upright position is resumed and the maneuver repeated three or four times.

D. The above exercise should be increased gradually so that in two or three weeks the number of stretches is increased to ten or fifteen twice daily.

E. *This exercise should not be done* if there is pain in the sciatic nerve, since to do so will only increase the pain in the nerve.

The results in many instances have been highly satisfactory and have given relief from low back pain and have improved postures. The stretching exercises must be carried out over a period of several weeks or months or until the contractures have been relieved. When this has occurred, the simple exercises to improve the condition of the muscles that support the spine must next be prescribed. The principal muscles which support the low back are not the erector spinae muscles but are the abdominal and gluteus maximus muscles.

Indication for the Operative Relief of Fascial Contractures.—(1) meralgia paresthetica, (2) sciatica of long standing, (3) sciatica plus lame back, (4) chronic lame back, (5) after unsuccessful lumbosacral fusion, and (6) failure after fasciotomy on the opposite leg.

1. *Meralgia Paresthetica.*—This is due to irritation of the lateral femoral cutaneous nerve. It is seen in fat people and in those who have contracted fasciae latae and is sometimes associated with pain along the sciatic nerve.

2. *Long-Standing Sciatic Pain.*—In these cases it is important to rule out any pathologic condition in the spinal canal or in the neural foramen or tumors of the sciatic nerve itself before even considering fasciotomy. Having done this and finding that the fascial structures of the thigh are contracted, then, and only then, should section of the fascia and iliotibial band be considered.

3. *Sciatica Plus Lame Back.*—The same holds true when there is a chronic low back disability associated with sciatic pain.

4. *Chronic Lame Back.*—In these cases where there are symptoms and physical signs of a chronic lame back and where there are no indications of local pathologic changes in this region and where the fascial contractures are prominent, one is justified in relieving the fascial contractures. Both sides must be done. The results of fasciotomy in these cases are not seen immediately because, due to the operative release of the tight fascial bands, the contours of the back are changed and a readjustment of the structures in the lumbosacral region takes place. When such readjustment is going on, the lumbar spine is slowly restored to normal. In this restoration process the ligaments, fasciae, and muscles in the lumbar region are slowly stretched out. As a result of this severe stretching, these structures pull at their bony attachments. This pull is more than apt to be painful, hence the low back symptoms continue until the stretching process is completed.

5. *Pain in the Back Following Unsuccessful Fusion.*—If a patient complains of backache after his spine has been fused, the pain may be due to the contracted fascial structures pulling on the low back. If this be so, then the contractures must be relieved. If the back has been fused in the lordotic position giving a permanent fixed lordosis, operation on the contracted fascial bands probably will not relieve the backache, since it is fixed for all time in the position of discomfort.

6. *After Failure of Fasciotomy on the Opposite Leg.*—In those patients whose sciatic pain has not been relieved by a unilateral successful fasciotomy, operating on the opposite side often will give relief if there are fascial contractures on that side also. It is wiser to allow some time to elapse, however, before proceeding with the second operation; first, because the nursing care of a unilateral fasciotomy is more simple when sciatic pain is present and, second, the pain may disappear in ten or twelve weeks. If the pain does not disappear and if there is marked contracture of the fascia, the mechanical effect is to tilt the pelvis toward the contracted side by its lever power, thus increasing the apparent adduction on the operated side. The result of this opposite tilt of the

pelvis is to produce spasm in the gluteus maximus by stretching or because for the same reason the position of adduction allows the muscular structures at the posterolateral aspect of the pelvis and hip to compress the nerves in that region and thereby irritate them.

Operative Treatment.—It has been stated that division of the fascia lata and the iliotibial band as advocated does not relieve lame backs. The writer's experience is quite to the contrary.

Division of the fascia lata and iliotibial band has been described in preceding articles[1, 2] but some modifications in technique have been developed since the early cases were done. If the anterior part of the fascia in the region of the sartorius and rectus femoris is tight, it must be divided. All of the contracted intermuscular septa must be found and divided, particularly the thin septum on the medial aspect of the gluteus maximus. In other words, a thorough division of all the tight structures will relieve the contractures that can be clinically demonstrated by the straight leg raising, the leg flexion on the thigh, and the abduction tests. When this happens there will be no place for trying to change the operative procedure as outlined by trying to insert modifications. Surgeons too often try to modify, forgetting that the originator of a special technique has already tried all of the modifications in working out his problem and has dismissed them because they either did not work or complicated a simple procedure.

One question often asked is this: "Do you suture the divided fascia?" The answer is no, since to do so would only reproduce the original picture and the result would be a failure.

Another question asked is: "How does the fascia heal?" The answer is that the fascial gap will be closed by fascial tissue. Those who did Soutter fasciotomies in the past and who had occasion to do them again for recurrences always noticed that the fascial gap had healed over perfectly with tissue that resembled the original fascia.

Postoperative Care.—The patient is returned to bed from the operating room. A pillow is placed under the knee of the operated leg and a cradle over the feet. In unilateral cases the patient is allowed to sit up in bed the next day if there is marked relief of the sciatic pain. If there is not much relief, the patient should be kept recumbent and comfortable until relief begins when he is allowed to sit up in bed and then is allowed to sit up in a chair. Most of the patients get so much early relief from their pain that it is permissible to allow a little walking as early as the third or fourth day. In those cases where double fasciotomy has been done for the chronic lame back, the patient should be kept in bed for from seven to twelve days. At the end of this time he may be allowed to sit and to walk.

Physical therapy is begun as soon as the sciatic pain or lame back symptoms have disappeared. This treatment consists in baking, gentle

massage, and exercises to improve the power of the corset muscles, which are the gluteus maximi and the abdominal muscles. Particular attention must be paid to the gluteus maximus on the side where there has been sciatica, since it is always atrophied and sometimes to a considerable degree. If this atrophy is not overcome, pain is apt to occur along the sciatic nerve when the patient sits down because there is not enough protective padding between a nerve that has been irritated for a long time and the seat on which the patient sits.

Results.—The results of treatment of lame back conditions and sciatic pain are good enough to warrant a continuance of efforts to relieve fascial contractures in the leg and back when such contractures are present. In a paper published last August,[3] a report was given showing the results in cases of fasciotomy. The percentage of those relieved of sciatic pain was 75.

The time before relief occurs varies from immediately to six months. However, most of the patients are relieved in from four to six weeks. The relief obtained in chronic lame backs after fascial division is usually slower than in sciatic pain cases for the reason that it takes a long time for the chronic back to become readjusted to its normal physiologic position. This is due to the fact that ligaments, fasciae, and muscles have become adaptively shortened and it takes time for them to become stretched.

The lame backs which are not severe are often relieved by the side stretching exercise described above. These patients improve under general physiotherapy measures plus stretching of the contracted leg muscles and fascial structures. A good percentage are completely cured.

Postural disturbances with fascial contractures improve more rapidly if the contractures are stretched out first. This stretching relieves the abnormal pull on the pelvis and is transmitted to the whole spine at the lumbosacral junction and the spine can then assume its normal physiologic contours. After this has been accomplished, it is time to begin a system of exercises to strengthen the muscles that hold the spine in a normal position. In other words, the deforming factors must be removed since the deformity, which is more or less permanent, increases the burden on the musculature that is trying to maintain a posture against a contracture.

SUMMARY

1. It has been demonstrated that fascial contractures occur at the thigh-pelvic junction.

2. There is a definite relation between muscle and fascial contractures of the leg and the mechanics of the whole spine.

3. There is a definite relation of fascial contractures to sciatic pain in some cases of sciatic pain.

4. It must not be assumed that contracted fasciae are responsible for all cases of sciatic pain.

5. In examining patients with lame backs or bad posture, it is necessary to determine the relation of the lower extremity to the lumbosacral and sacroiliac joints; i.e., whether there is limitation of straight leg raising, limitation of leg flexion on the thigh with the patient prone, or positive abduction test.

6. Bad postures and lame backs are more promptly relieved if fascial contractures are disposed of early.

7. Operative treatment for sciatic pain should not be advised until a proper diagnosis has been made. It should be postponed until all other conservative measures have failed.

8. Fasciotomy is a relatively simple operation but its usefulness is limited to a selected type of case.

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SURGICAL TREATMENT OF AFFECTIONS OF THE LUMBOSACRAL AND SACROILIAC JOINTS

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LOW back pain, one of the most common complaints, arises from a number of causes, the degree of disability which it produces varying from slight discomfort to complete invalidism of the patient. In most instances the pain originates in the vertebral column and pelvic articulations and in a lesser number the symptoms are due to disease involving abdominal or pelvic organs not connected with the spine but producing pain referred to the low back region. The second group or those in which the pathology is external to the spine must always be excluded before making the diagnosis; however, this discussion is confined to those cases in which the symptoms are due to localized affections of the lumbosacral and sacroiliac articulations.

Certain anatomic facts upon which low back pain depends deserve mention. In the male locking of the sacroiliac joint is sufficient to prevent all but the slightest movement, while in the female the articulating surfaces of the pelvic bones are smoother, the muscles are weaker, and the joint is capable of a greater range of motion. Women, therefore, are more liable to sacroiliac strains than are men. The fifth lumbar nerve root is the largest nerve root; it passes through the smallest intervertebral foramen and may therefore be compressed and irritated by any condition which reduces the lumen of the foramen. The root of the fifth lumbar nerve lies directly anterior to the articular facets between the last lumbar vertebra and the sacrum, and the lumbosacral plexus passes anteriorly to the sacroiliac joint. Inflammatory exudates within either of these joints may easily involve the nerve roots, resulting in sciatic pain. Also the fourth and fifth lumbar nerves possess longer funicular portions than the other spinal nerves and this part, unlike the intraspinal portion, does not lie within the arachnoid sheath and is not bathed in cerebrospinal fluid. Thus the funiculus is exposed more readily to external mechanical influences, and, because of the surrounding venous plexus, it is affected by congestion and stasis in the neighboring tissues. The transition from the freely movable vertebral bodies to the relatively immobile pelvic joints produces an area of mechanical instability. Furthermore, congenital anomalies are prone to occur at the transitional regions of the spinal column.

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Etiology.—The causative agents which affect the spine and cause low back pain may be enumerated in order of frequency as follows: (1) infection, (2) congenital anomalies, (3) trauma, (4) postural defects, and (5) tumors. Infectious arthritis with or without arthritic changes demonstrable by the roentgen ray is undoubtedly the most common cause of low back pain. Adults are affected by arthritis more frequently than children and males more often than females. The joints involved may be one or both sacroiliac joints, the articular facets or the intervertebral disk between the last lumbar vertebra and the sacrum. Infectious spondylitis or osteoarthritis is an inflammatory disease associated with pyogenic foci of infection. The disease process does not lead to suppuration but in healing degenerative changes occur in the hyaline cartilage of the joint and the adjacent bone structure becomes eburnated. Inflammation of muscles or fasciae, producing myositis or fibrositis of the sacrospinalis, gluteus maximus, or gluteus medius muscles, is frequently present. Calcification of the iliolumbar and sacroiliac ligaments at times may be demonstrated by the roentgenogram. The nucleus pulposus may be ruptured, causing narrowing of the intervertebral space with erosion of the bony surfaces. Tuberculous or other destructive processes are a more serious, but fortunately a less frequent, cause. Tuberculosis of the spine and sacroiliac joints, formerly common in children or young adults, is now becoming rare.

Congenital anomalies, such as enlarged transverse processes and spina bifida occulta, sagitally placed articular facets, and numerical variation of the spinal segments, are not in themselves a cause of low back pain, but such structurally weakened backs are more susceptible to strain. Of the many anatomic variations of the lumbosacral region, the most important ones are abnormal inclination of the pelvis with shearing stress at the lumbosacral junction, congenital lack of bony fusion between the vertebral pedicle and lamina which results in spondylolisthesis and impingement of a sacralized transverse process. Other more gross defects, such as hemivertebra, wedging of the vertebral body with extreme distortion and displacement, may be regarded as definite causes. Tumors within or without the spinal canal should be borne in mind and in persons past middle age malignancy should always be considered in the differential diagnosis.

Under the heading of trauma are included sprains, strains, fractures, and dislocations. A sprain is a distinct rupture of a ligament accompanied by extravasation of blood and subsequent scar formation. Trauma, of course, may be an instigating factor whenever there is a defect in the skeleton from any cause, but it should be considered as a primary factor only when there is a definite relationship between the injury and the onset of symptoms. Even in those with a definite history of trauma, the possibility of associated infection from a distant focus must be considered especially in patients past the age of thirty-five years.

A number of industrial and other cases of low back pain are probably due primarily to strains of the joints of the lower spine. The disability is prolonged frequently by delay in securing proper treatment, toxic absorption or irritation from distant foci of infection, and faulty posture.

Postural defects with relaxed musculature placing additional strain on the ligaments may be a predisposing factor. The association of other static anomalies, such as flatfoot, kyphosis, and visceroptosis, is a common finding. In no instance should faulty posture be accepted as the sole cause of the symptoms, although it is well recognized that lumbosacral pain may be aggravated by such conditions, especially increased lumbar lordosis. In this type of individual operative methods are rarely advisable and treatment should be directed mainly toward improvement in body mechanics. Scoliosis of both functional and structural varieties predisposes to strain of the lumbosacral joint. Only in scoliosis of extreme degree are symptoms present during childhood and, as a rule, pain resulting from muscular and ligamentous strain, fatigue, or pressure upon nerve roots is not experienced until maturity.

Symptoms and Diagnosis.—The symptoms may come on suddenly or gradually. The chief symptoms are (1) pain either localized or referred, (2) limitation of spinal movements by muscle spasm, and (3) characteristic attitudes, such as flattening of the lumbar spine and sciatic scoliosis, which are assumed by the patient reflexly or subconsciously in order to protect and immobilize the painful area. The examination of the spine should be made with the patient in three positions, standing, sitting, and lying. When the patient is standing, muscular rigidity is more pronounced and the characteristic attitude is more apparent. Motion is limited to a greater degree when the patient is erect; when the patient is in the sitting position, tension of the hamstring muscles on the pelvis is relaxed and forward bending is less restricted. In the recumbent position passive movement of the spine may be carried out to practically the normal range. Localized points of tenderness may be demonstrated by pressure over the lumbosacral or sacroiliac joints, the insertion of ligaments, or over the sciatic nerve. The examination must be systematic and carefully made to localize the exact site of pathology. Differentiation between lumbosacral and sacroiliac involvement may be facilitated by one of several tests. None of the signs is positively diagnostic and all are rarely present in a single case.

With the patient on his back, the affected leg is raised with the knee extended. As pointed out by Goldthwait, pain is often present on this manipulation in affections of the vertebral column. The exact time of the occurrence of pain is a valuable point in the differentiation of lumbosacral and sacroiliac lesions. One of the examiner's hands is placed under the lumbar spine, the other hand grasps the leg and flexes the hip; if pain is experienced by the patient before motion is detected in the lumbar spine, sacroiliac involvement is indicated. If the patient

does not complain of pain until there is movement in the lumbar spine, the affection is in all probability in the lumbosacral joint. Compression of the iliac crests is also a valuable diagnostic procedure. The patient lies on the affected side and the examiner presses downward firmly over the opposite iliac crest; if the sacroiliac joint is diseased, pain is felt in the affected joint. A variation of this sign, though not so reliable, is direct backward pressure over the pubic bone. Gaenslen has described a diagnostic maneuver which he believes is useful in differentiating between lumbosacral affections and between right and left sided sacroiliac lesions. The patient, lying supine, flexes one knee and hip acutely. The patient is then brought well to the side of the table and the opposite thigh is hyperextended slowly by the examiner with gradually increased force. As a result of the impaired ligamentous support on the affected side, this rotating force causes abnormal mobility accompanied by pain, either local or referred, in the region of the sacroiliac joint on the side of the lesion.

Roentgenograms should be taken routinely in both anteroposterior and lateral positions; semilateral views may be of value in demonstrating lesions of the articular facets. The roentgenograms are by no means conclusive. In many patients with definite symptoms the roentgenographic examination is negative; also, there are instances of positive findings which have no bearing on the existing symptoms. Actual roentgenologic evidence is a late manifestation, but, in a large percentage of the cases requiring surgical fusion of the lumbosacral and sacroiliac articulations, arthritic lesions may be demonstrated by routine physical and roentgenographic examinations. The joint space becomes narrowed, the articular surfaces eroded, the underlying bone condensed, and nature attempts to heal the process by the formation of bony bridges across the joint.

Treatment.—The patient who complains of low back pain presents a diagnostic problem, the solution of which requires careful examination and, frequently, treatment by specialists in various fields of medicine and surgery. The symptoms often may be relieved by appropriate measures to immobilize and stabilize these joints. For the acute lesions, adhesive strapping, rest on a firm bed, traction, plaster of Paris casts, spinal braces, and physical therapy are indicated. For the more chronic lesions, it is advisable to correct any postural defect by exercise, to remove all foci of infection, to promote elimination, to regulate the diet, and to support the spine by a brace or corset in addition to physical therapy.

Ankylosing operations on the spine are required frequently for persistent cases of lumbosacral and sacroiliac pain which have not responded to thorough conservative measures. The purpose of surgical treatment is to immobilize the affected joint or joints by an internal bony splint.

The classical operative procedures for fusion of the spine devised by Albee and Hibbs are well known and are well adapted for lesions in the lumbosacral joint. Operations for fusion of the sacroiliac joints have been described by Smith-Petersen, Gaenslen, Campbell, Picque, Verrall, and Chandler. The theory underlying each of these procedures is anatomically and physiologically sound and it has been proved clinically as well as experimentally that by either of the methods osseous fusion of the sacroiliac joint may be secured. The operation devised by Campbell for fusion of the sacroiliac joint has the advantage of being entirely extra-articular and thereby avoids the danger of relighting a latent infection in the joint which is a most serious complication; especially is this true in tuberculosis of this articulation as the resulting secondary infection may prove fatal. The procedure is as follows:

Extra-Articular Technique for Fusion of the Sacroiliac Joint.—A general anesthetic is employed, nitrous oxide being preferred. The patient is placed prone on the operation table with sand bags under the shoulders and a small pillow under the pelvis in order to permit free respiratory excursion of the thorax and abdomen. The field of operation is prepared with the antiseptic chosen by the surgeon and is draped with sterile linen. An incision is made over the outer lip of the crest of the ilium along the posterior one-third or one-half of the ilium to the posterior inferior spinous process. Towels are clamped to the wound margins and the incision is carried down to the bone. The periosteum is incised and elevated for a considerable distance, and the posterior portion of the dorsum of the ilium is exposed. The crest of the ilium is dissected free of fibrous tissue and the adjacent posterior surface of the sacrum is denuded of muscles and ligaments. A portion of the iliac crest, approximately 3½ to 4 inches long, is removed with an osteotome and preserved. The inner surface of the overhanging portion of the ilium and the adjacent posterior surface of the sacrum are scarified, thus forming a raw gutter parallel with the sacroiliac joint. The walls of this gutter are formed by the posterior surface of the sacrum and the inner surface of the ilium posterior to the sacroiliac joint. Into this space is placed the graft from the iliac crest. Multiple chips of bone as additional grafts are then secured from the dorsum of the ilium and placed into the gutter until the space is well filled. The bone fragments are wedged firmly into the gutter using a blunt-ended instrument as an impactor. The wound is closed in layers.

Technique for Combined Fusion of Lumbosacral and Sacroiliac Joints.—When desirous of fusing the lumbosacral articulation in addition to the sacroiliac joint, the posterior aspect of the transverse process of the fifth lumbar vertebra may be exposed and denuded through the same incision. The osseous graft from the crest of the ilium is placed into the gutter, already described, but extended upward to approximate the denuded surface of the transverse process of the fifth lumbar vertebra.

This extension to include the fifth lumbar vertebra has been applied more recently in a number of cases with distinct success. If preferred in some cases, a curved or convex graft from the crest of the ilium may be transplanted into the spinous processes of the lumbosacral region after the manner of Albee. A second skin incision in the midline is required for this purpose.

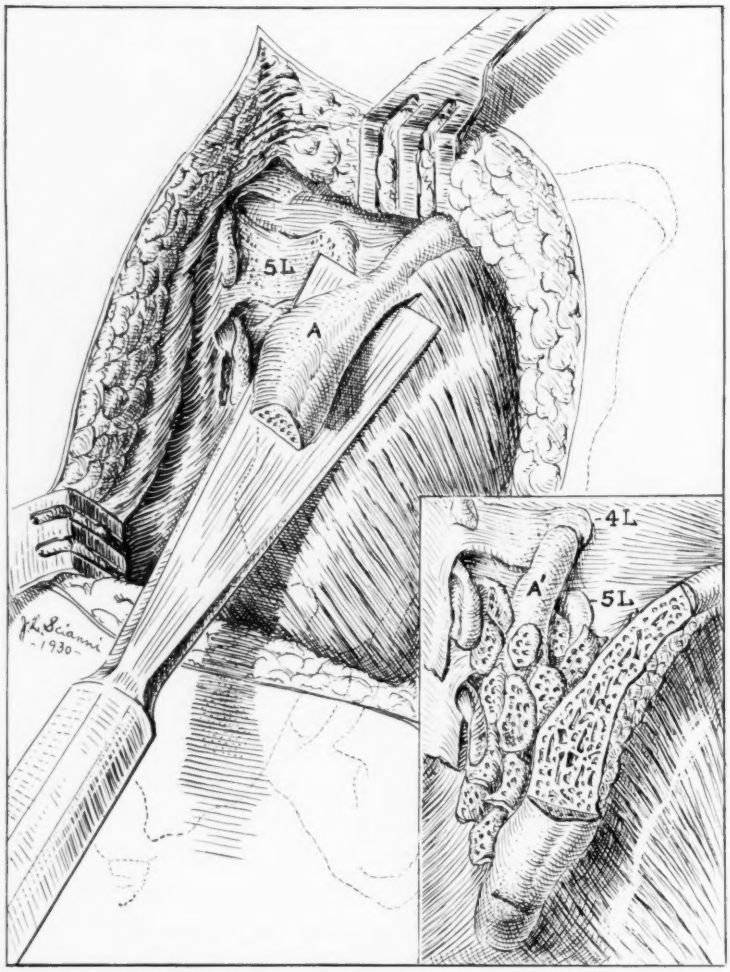

Fig. 1.—Exposure for extra-articular fusion of the sacroiliac joint. The posterior portion of ilium, *A*, is removed with an osteotome. Insert shows removed portion of ilium, *A*, lying on denuded surface of sacrum and transverse processes of fourth and fifth lumbar vertebrae. Multiple bone chips are placed into the denuded gutter.

Technique for Fusion of the Lumbosacral Joint.—When the lumbosacral region is involved primarily, a bone graft operation modified from the technique of Albee is employed. Because of the normal lordosis at the lumbosacral junction, the spinous processes of the lower lumbar vertebrae and the sacrum are not in the same plane. A straight tibial graft, therefore, is not suitable and a curved or angulated graft should

be employed. Through a curved incision the spinous processes are exposed; the supraspinous and intraspinous ligaments are incised longitudinally, and the spinous processes are split in half by a sharp osteotome almost to the neural arches; one-half of each spinous process is fractured completely at its base and displaced laterally. The graft bed thus presents a median longitudinal gutter extending from the spinous process of the second or third lumbar vertebra to the spine of the third

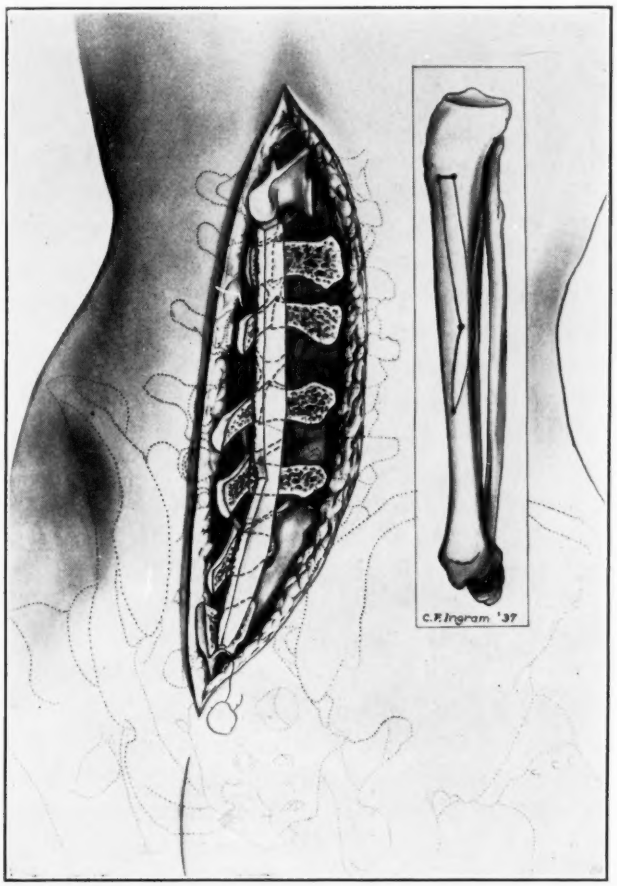

Fig. 2.—Ankylosis of lower lumbar vertebrae and sacrum, by means of a curved graft which is shaped to accommodate the normal lumbosacral lordosis. Insert shows the removal of the curved graft from the tibia.

or fourth sacral segment into which a graft of sufficient length may be placed. Flaps of bone may be turned upward and downward from the adjacent laminae to give additional support. As the spinous processes of the sacrum are too small to permit sufficient bony apposition, a groove or flap must be made into the posterior surface of the sacrum for the reception of the massive curved graft.

A tourniquet is applied to the thigh while the tibial graft is being removed. The anteromedial surface of the tibia is exposed by a straight

or curved incision. The periosteum is reflected from the bone and the muscles are stripped subperiosteally from the middle portion of the shaft along the posteromedial border. Small drill holes are made into the flat surface of the tibia to outline the curved graft which is angled or curved with the convexity posteriorly, the apex of the curve consisting of a portion of the posteromedial surface of the tibia. By this method the tibial crest is not violated, thus preserving the strength of the tibia for early weight-bearing. A graft from 6 to 8 inches long is removed with the motor saw. The upper end of the graft is made blunt as it will be applied to the spinous processes of the lumbar vertebrae and will be covered by soft tissue. The lower end of the graft which lies over the sacrum is pointed since this portion will be almost subcutaneous. A graft so shaped will fit snugly into the ordinary lumbosacral angle, but, if the lordosis is so great that conformation cannot be secured by such a graft, an osteoperiosteal graft may be employed with as much bone as possible being left attached to the periosteum. Cancellous bone from the tibial condyles is placed about the graft to promote osteogenesis. The supraspinous ligament is sutured over the graft and the wound closed. Considerable time may be conserved by having two operating teams—one to prepare the spine for reception of the graft and the second to remove the graft from the tibia.

Postoperative Care.—After completion of the operation, the patient is placed on a Bradford frame for a period of about two weeks. When the wound is healed and the skin sutures have been removed, a plaster of Paris cast is applied extending from the axillae to the knee on one or both sides. The cast is worn for six weeks and when it is removed the patient is fitted with a spinal brace with a sacroiliac belt which is worn for at least one year or until all symptoms have subsided.

Analysis of Cases.—In 1930 my associate, Dr. Willis C. Campbell, reviewed the end-result in 63 patients on whom fusion of the lumbosacral and sacroiliac joints had been performed. Since that report, 51 additional patients have been operated upon, making a total of 114 fusion operations for affections of the lumbosacral and sacroiliac joints. The final diagnosis in these cases was as follows: low-grade infectious arthritis and spondylitis in 53 cases; tuberculosis, 22 cases; trauma, 9 cases; congenital anomalies, 19 cases, of whom 13 were spondylolisthesis; benign tumor, 1; and cord tumor, 1. Of the entire group 67 were males and 47 were females. The age of most of the patients varied from 25 to 50 years. Three of the patients were under 10 years of age, the youngest being 5 years. The oldest was 53 years.

The patients with infectious arthritis comprised the largest group, and it should be emphasized that arthritis is the most frequent cause of low back pain. A small number of cases with infectious arthritis gave a definite history of trauma, while in a somewhat larger group the history of injury was more uncertain and indefinite. Trauma should be

considered as the primary factor only when there is a direct relationship between the occurrence of injury and the onset of symptoms. Of the infectious group, males were slightly more numerous than females. The majority of the patients in this group were in the third and fourth decades of life. Patients with osseous tuberculosis are as a rule younger than those with infectious arthritis.

In the traumatic group there were 9 patients. In 5 gross fractures in the vertebrae or pelvis were recognized; in 4 instances, while traumatic lesions were not demonstrated, the injury and subsequent symptoms were so severe and persistent that a diagnosis of traumatic arthritis was justified. Of the patients with spondylolisthesis, 11 were males and 2 were females. One patient had a benign tumor and in 1 patient in whom lumbosacral and sacroiliac arthrodesis was done a tumor of the spinal cord was recognized a few months later and was removed.

The roentgenograms demonstrated definite abnormality, either atrophic, destructive, or traumatic lesions, or congenital anomaly in one or more articulations in 103 cases. Repeatedly negative roentgen findings were present in only 11 of the cases. This fact illustrates the value of the roentgen ray as an aid in diagnosis and also illustrates the care with which the patient who complains of low back pain should be selected for surgical treatment. Of the 114 patients, there were 93 in whom operative measures were applied for fusion of the lumbosacral articulation; 63 of these were fusion of the lumbosacral joint alone, and 30 were fusions of the lumbosacral articulation combined with fusion of one or both sacroiliac joints. In 21 cases the sacroiliac articulations were fused. In fusion of the lumbosacral joint the Albee type of graft was employed in 48 cases. In the remainder an osteoperiosteal graft was employed or the graft from the ilium was placed in contact with the transverse processes of the lumbar vertebrae as has been described. In fusion of the sacroiliac joints the Smith-Petersen procedure was used in 3 cases and the remainder were fused by the Campbell extra-articular method.

RESULTS

Since the clinical course and prognosis in tuberculosis varies so widely from that of other types of arthritis, a separate classification is justifiable. The results of operative fusion are summarized in the accompanying tables, Table I representing the nontuberculous cases, and Table II those

TABLE I

RESULTS IN NONTUBERCULOUS CASES

	EX-CELLENT	GOOD	POOR	DIED	UN-KNOWN	TOTAL
Sacroiliac (alone)	8	3	2	0	2	15
Lumbosacral and sacroiliac	11	4	9	2	3	29
Lumbosacral (alone)	25	7	4	0	12	48
Total	44	14	15	2	17	92

TABLE II

RESULTS IN TUBERCULOUS CASES

	EX-CELLENT	GOOD	POOR	DIED	UN-KNOWN	TOTAL
Sacroiliac (alone)	2	0	2	2	0	6
Lumbosacral and sacroiliac	0	0	0	0	1	1
Lumbosacral (alone)	6	5	1	3	0	15
Total	8	5	3	5	1	22

with tuberculosis. By excellent results is meant complete relief. In results classified as good, pain of some degree persisted but there was partial and permanent improvement. Poor results indicate no relief of symptoms following operation. There were 7 deaths, a mortality rate of 6 per cent. This statement deserves explanation. Two of the 92 non-tuberculous patients died of operative shock, an operative mortality of 2 per cent. These 2 operative deaths occurred in the earlier series of cases in attempted fusion of the lumbosacral and both sacroiliac joints at one operation. Since these fatalities, the combined operation has not been employed except in very robust individuals. There is little postoperative shock associated with the fusion of the lumbosacral and one sacroiliac joint or bilateral fusion of the sacroiliac joints by the methods described. In less robust patients, however, it is advisable to perform the multiple fusions at separate operations. In the tuberculous cases there were no operative deaths. Five patients with tuberculosis died subsequently as the result of complications. The unknown group includes patients operated upon recently as well as those not followed sufficiently long to determine the ultimate result. Fifty-eight, or 77 per cent, of the non-tuberculous cases in which the final results are known obtained complete relief of pain or were markedly improved following operation. Thirteen, or 61 per cent, of the tuberculous patients were similarly benefited.

CONCLUSIONS

Low back pain is a common symptom of affections in the lumbosacral and sacroiliac articulations. Conservative treatment is effective in relieving the majority of patients, but in a small percentage in whom symptoms persist in spite of conservative measures surgical fusion is recommended.

The technique for extra-articular fusion of the sacroiliac joint and a method for ankylosing the lumbosacral joint are described.

Fusion operations are advisable in all cases of tuberculosis of the lumbosacral and sacroiliac articulations, in those nontuberculous cases with persistent symptoms and definite abnormalities demonstrated by the roentgenogram, and in those cases in which symptoms persist after conservative treatment, even though the roentgenogram will demonstrate no pathologic change.

The operation should be performed with precision; not over one hour should ever be consumed for the procedure. If the operative time is unduly prolonged, shock with an increase of mortality may be expected.

In a selected group of cases definite improvement may be expected in more than 75 per cent following fusion operation.

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THE FUNCTION OF THE SPLEEN IN THE RETARDATION OF SHOCK FROM HEMORRHAGE

An Experimental Study

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BY COMMON surgical tradition, the removal of the spleen offers no serious handicap to the individual after a period of time has passed. It is known that within a few months the immediate postsplenectomy qualitative changes in the blood, such as anemia and increased resistance of the red cells to hemolysis, disappear, and the blood becomes practically normal.[1, 2] It is believed that other parts of the reticuloendothelial system take over these functions. There is, however, a function of the spleen that these other tissues cannot assume; namely, the so-called "reservoir function," which has to do with splenic response to physiologic demands for more blood. By reason of its capacity to change volume and its "bypath" position in the circulation,[1, 3] the spleen is capable of storing blood and delivering it promptly to the circulation when there is need. So far as is known, this function remains in default after splenectomy.[1] The present study presents an attempt to determine how important this defect may be in delaying the development of shock from hemorrhage in the dog.

Experimental studies have demonstrated the contraction of the spleen and the results of such contraction under circumstances demanding either an increase in blood volume or more efficient oxygenation. Barcroft's[3] important observations showed that the spleen contracts under active exercise, mental agitation, oxygen want, anesthetics, and hemorrhage. Moore[4] demonstrated splenic contraction in traumatic shock. Binet, Cardot, and Williamson[5] found that the marked elevation of red cell count that occurs during asphyxia from compression of the trachea is absent after splenectomy. Barcroft[3] showed that intact animals poisoned by carbon monoxide survived longer than splenectomized animals; and De Boer and Carroll[6] ascribed the longer survival of the normal animals to the delivery to the circulation of unpoisoned red cells from the spleen which reduced the ratio of carbon monoxide hemoglobin to oxyhemoglobin in the circulating blood. Eppinger and Schürmeyer,[12] in confirming certain of Barcroft's observations, concluded from experiments on animals that other blood reservoirs be-

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sides the spleen must exist. In addition, with exercise and increased environmental temperature, a single splenectomized patient revealed a definite increase in blood volume which, however, was somewhat less than that found in normal subjects.

The experiments most pertinent to the present study are those of Viale,[7] who studied the effects of rapid hemorrhage on the blood pressure of animals with and without the spleen. He found that the spleen protects the animal from the immediate drop in pressure following hemorrhage. He withdrew from a dog 19 c.c. per kilogram in 37 seconds. The systolic blood pressure was 102 mm. Hg immediately afterward and 124 mm. Hg one minute later. With the spleen out of the circulation, this prompt recovery did not occur. For instance, he withdrew 4 c.c. per kilogram in 24 seconds with no change in blood pressure. The splenic pedicle was then ligated and after an additional 2.5 c.c. per kilogram were removed (a total of only 6.5 c.c. per kilogram), the blood pressure promptly fell to 10 mm. Hg with no recovery. Barcroft[3] found that there is no decrease in the circulating blood volume during the early stages of repeated small withdrawals of blood and that the amount withdrawn is approximately equalled by the amount delivered by the spleen.*

A practical surgical question that remains unanswered by these observations is whether the splenectomized patient can actually meet occasional severe demands for an increase of blood as well as the normal individual. No warning has been found in the surgical literature that the splenectomized patient should be approached surgically with caution. Except for Viale's[7] report, there is no study of the effect of marked blood loss on the splenectomized animal as compared with the normal animal.

METHODS

Dogs, without selection of breed, sex, or size, were employed. They were kept on a stock laboratory diet until about 36 hours before each experiment. The period of starvation was adopted to exclude in the controls changes in spleen volume associated with active digestion. Water was allowed *ad libitum*. Forty-six animals were observed, of which one-half were splenectomized and the remainder retained as controls.

Splenectomy was performed under anesthesia, with aseptic precautions and silk technique. A careful search was made for accessory spleens. A recovery period was maintained before subjecting the splenectomized dogs to further experimental procedures. The interval varied from 25 days to 61 days with an average of 36 days. Before the splenectomized animals were further studied, the absence of anemia was demonstrated.

*For consideration of the mechanism of splenic contraction, see Wiggers,[1] Barcroft,[3] Izquierdo and Cannon,[8] Viale,[9] and Viale and Soncini.[10]

The arterial blood pressure of both control and splenectomized dogs was observed during measured blood loss. Intraperitoneal sodium amytal anesthesia was employed. The arterial blood pressure was recorded by a continuous kymographic tracing from a cannula in the femoral artery. Blood was withdrawn from the opposite femoral vein at a uniform rate of 5 c.c. per kilogram of body weight every 5 minutes, until the dog was in profound shock or death ensued.

RESULTS

Differences between the control and the splenectomized animals were found both in the behavior of the arterial pressure immediately after each withdrawal of blood and in the effect of the total loss.

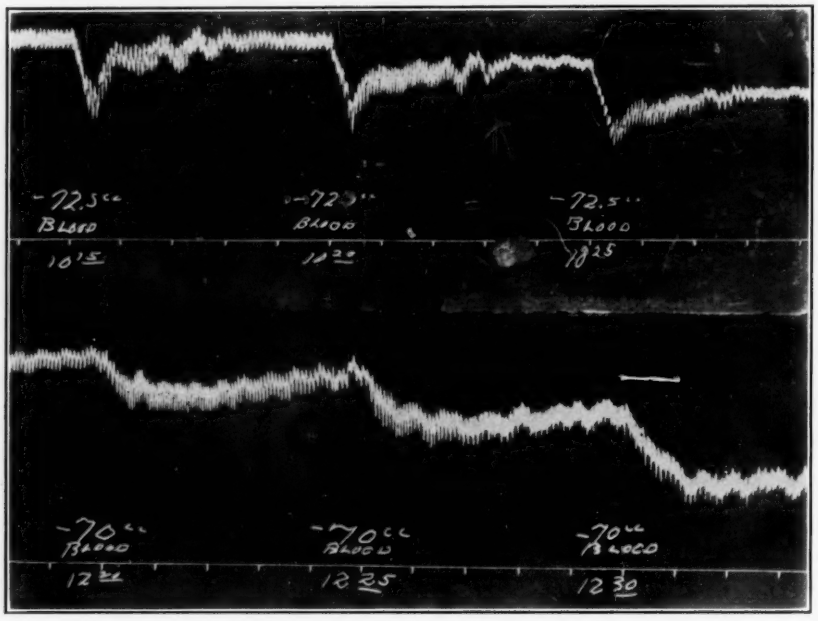

Fig. 1.—Arterial blood pressure curves from typical animals. The upper tracing represents the tracing from a control dog; the lower, from a splenectomized dog. Note the failure of immediate partial recovery of pressure following each bleeding in the latter as compared with the former. This difference was not present in all tracings.

After each withdrawal of 5 c.c. of blood per kilogram from the intact animal, the consequent immediate drop of blood pressure is in part transient. In the course of a few heart beats the pressure climbs back to a level somewhat below the original level (Fig. 1, upper tracing). In the splenectomized animal, however, this recovery is not apt to occur. The pressure tends to remain at the level to which it has dropped with the withdrawal of blood. (Fig. 1, lower tracing.) Although this difference cannot be detected in every tracing, it is sufficiently frequent to confirm Viale's observations.[7]

The difference observed in the reaction of the splenectomized dogs to repeated bleeding as compared with the intact dogs is equally notable.

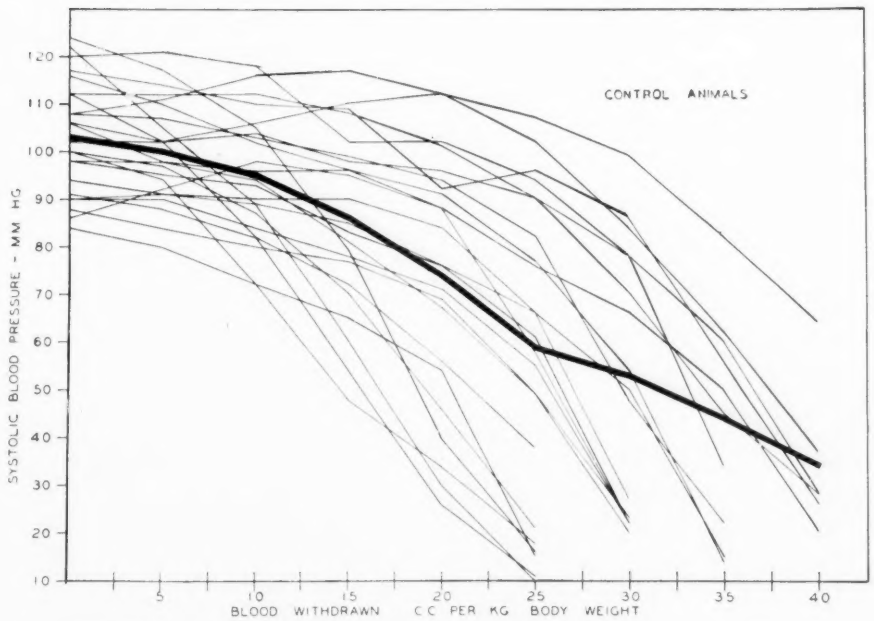

Fig. 2.—Control dogs. Individual and average arterial blood pressure curves during withdrawal of 5 c.c. of blood per kilogram of body weight at 5-minute intervals. Compare with Fig. 3.

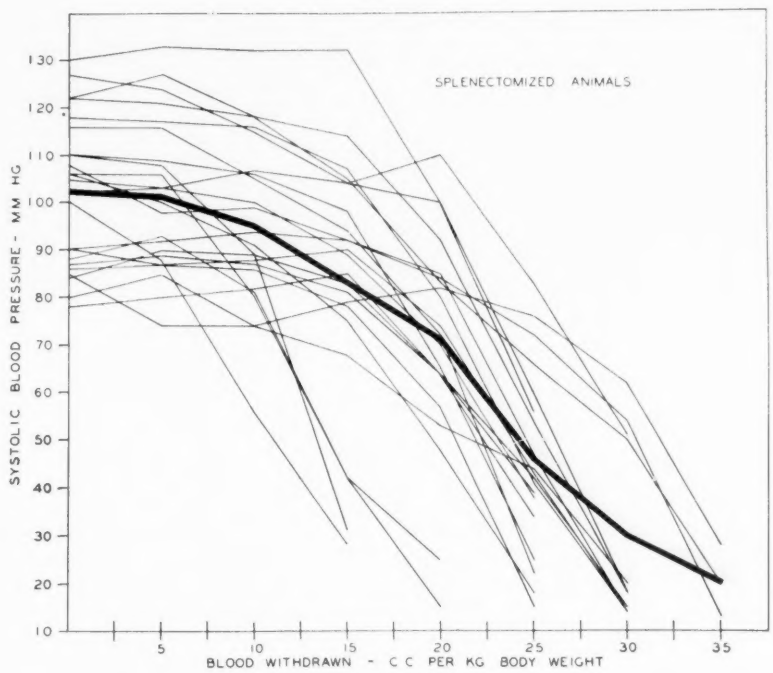

Fig. 3.—Splenectomized dogs. Individual and average arterial blood pressure curves during withdrawal of 5 c.c. of blood per kilogram of body weight at 5-minute intervals. Compare with Fig. 2.

The blood pressure curves tend to drop at a sharper angle, so that a shock level of 50 to 60 mm. Hg is more quickly reached and death tends to occur sooner (Figs. 2 and 3). There is, of course, considerable overlapping of the two sets of curves. Some individual splenectomized animals sustained hemorrhage better than some individual controls. It will be noted, however, that 4 animals in the experimental group succumbed before a single intact animal died; and that 6 controls lived after all splenectomized dogs were dead. It will be noted also that, when 30 c.c. per kilogram had been withdrawn, 16 (70 per cent) of the 23 controls were still alive, as compared to 11 (48 per cent) of the 23 experimental animals.

The difference of reaction is probably fairly presented in the comparison of the curves of the averages for each group (Fig. 4). The

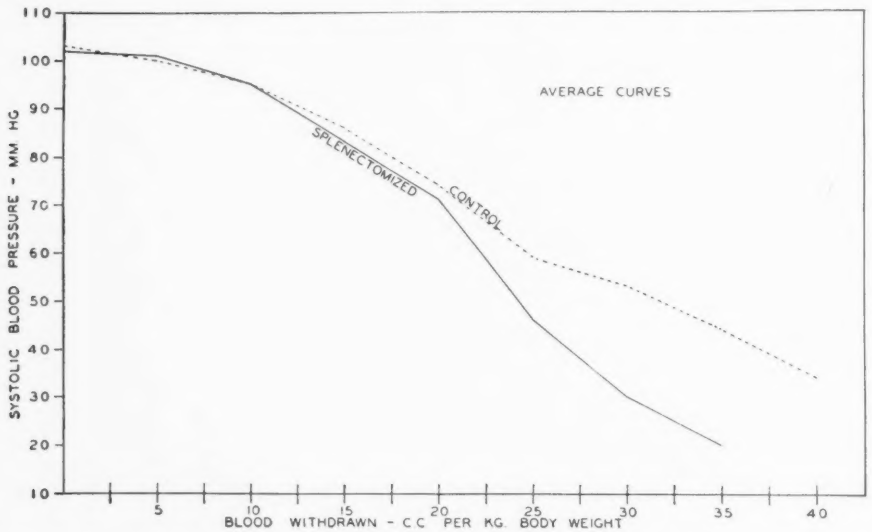

Fig. 4.—Comparison of average arterial blood pressure curves in control and splenectomized dogs (from Figs. 2 and 3).

average curve for the splenectomized animals drops more sharply than that for the controls and shock ensues earlier. When 30 c.c. per kilogram have been withdrawn, there is a difference of 23 mm. Hg in the average arterial pressures. In the converse sense, it requires the withdrawal of 31.5 c.c. per kilogram to drop the average arterial pressure in the intact animal to the level of 50 mm. Hg, as compared to only 24.25 c.c. per kilogram after splenectomy. In other words, apparently about one-third more blood loss is needed to produce profound shock when the spleen is present than when it is absent.

DISCUSSION

The present experiments indicate that the splenectomized dog sustains blood loss by repeated withdrawals less well than the intact dog.

From the physiologic evidence summarized earlier, this would be the logical and expected result. It is curious that the known physiologic facts in regard to the reservoir function of the spleen have not affected surgical thought. Holman[11] alone has considered this phase of the spleen's behavior in studying the temporary rise of blood pressure that follows the removal of the spleen, and especially of the enlarged spleen in the human being. It would be too much to assume that the present experiments can be carried over for direct application to the clinic. In the first place, it would be desirable to have them supplemented by studies at a longer interval after splenectomy to confirm the accepted opinion[1] that later compensations for the lost reservoir function are not developed. In the second place, there are differences in structure and relative volume of the spleen in man and in various laboratory animals, including the dog.

These experiments, however, should serve as a warning that a similar lack of protection from the effects of hemorrhage may be present in the splenectomized patient as has been demonstrated in the splenectomized dog. It may be suggested that the individual who has lost his spleen should be looked on by the surgeon as possibly a somewhat poorer risk for operation or accidental trauma on that account; and that the surgeon should be on the alert for an earlier development of shock than he would otherwise expect. If the figures given could be applied to man, rough calculation would show that a 70 kg. patient, who presents profound shock after the loss of about 2,000 c.c. of blood, would without his spleen withstand the loss of no more than 1,500 c.c. Certainly the surgical literature and surgical textbooks should cease to assume that the removal of the spleen definitely does not affect the chances of survival under all circumstances.

CONCLUSIONS

The splenectomized dog at a period less than two months after splenectomy sustains repeated withdrawal of blood (5 c.c. per kilogram of body weight at 5-minute intervals) less well than the intact dog. The arterial blood pressure curve drops more sharply; blood pressure recovery after each small bleeding is less apt to occur; the shock level is reached after less blood is lost; and death ensues earlier.

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AN EXPERIMENTAL STUDY OF OPERATIONS WHICH INVOLVE EXCLUSION OF THE PANCREATIC SECRETION FROM THE INTESTINAL TRACT, WITH SPECIAL REFERENCE TO THE POSSIBLE EFFECTS ON PROTEIN AND FAT DIGESTION AND ON THE METABOLISM OF THE LIVER CELL

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FOR both anatomic and physiologic reasons, malignancies arising in the ampulla of Vater, the second portion of the duodenum, the lower end of the common duct, and the duct of Wirsung present a common therapeutic problem in which many cases of carcinoma of the head of the pancreas might well be included. It is a problem, furthermore, which is encountered more often than is generally realized. Malignancies of the pancreas are relatively frequent, and even though malignancies of the ampulla of Vater are relatively infrequent, the evidence of Rolleston and McNee[1] and of others shows that one-third of the malignant growths of the extrahepatic bile ducts occur in this region, as do two-thirds of the malignancies of the duodenum.

Other considerations are also of interest. As Upcott[2] wrote in 1912: "There is probably no position within the body outside the central nervous system where a growth, while yet so small, is heralded by more widespread symptoms." Cooper[3] in 1937 wrote of "the dramatic early symptoms which facilitate diagnosis while the tumor remains localized." How much they facilitate diagnosis may be open to question, but of the importance of the symptoms they give rise to there can be no possible doubt. These symptoms are due to obstruction of the bile flow or of the pancreatic flow or of both, and they lead with incredible swiftness to hepatic insufficiency, pancreatic insufficiency, and the hemorrhagic diathesis which is one of the most frequent and most dangerous complications of obstructive jaundice of any origin. In other words, they are the manifestation of pathologic states which are compatible with life for only a limited period of time.

In all malignancies of this region the expectation of life is very short. Their brief duration is one of the notable features of carcinoma of the liver, carcinoma of the gall bladder, and carcinoma of the pancreas, as well as of all malignancies of the ampullar region. The average dura-

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tion of life in 47 collected cases of carcinoma of the ampulla of Vater in which the duration could be determined was, according to Outerbridge,[4] only 7½ months; in 23 cases it was less than 6 months and in 10 cases it was less than 3 months. Yet in only 25 per cent of his total series of 110 cases did death occur from malignancy per se, and in only 22 per cent of the whole series were metastases present at autopsy.

On the surface there are hopeful factors in this picture. Against the seriousness of the complications to which malignancies in this region give rise may be set the promptness with which they make themselves known. Against the swiftness with which the fatal end ensues may be set the apparently slow rate of metastasis. But the hopeful factors are more apparent than real. The disease may make itself known promptly, but even malignancies which manifest themselves early manifest themselves late, and metastases are absent not because the malignancy is of a mild character but because the end comes before they have had time to develop.

Because of the inevitably fatal character of carcinoma in this region there can be no doubt of the justification for surgery in any patient not obviously moribund. Because of the swiftly fatal character of the disease there can be no doubt of the justification for prompt surgery. No time must be wasted in attempts at hair-splitting diagnoses, if for no other reason than that the patient with obstructive jaundice of any origin presents an inherently poor surgical risk and a risk which increases the longer the jaundice endures. The necessary surgery is always formidable and may be lethal, for there are many factors which militate against success. Judd and Hoerner[5] list them as the insidious onset of the disease, the generally poor condition of the subjects, the relative inaccessibility of the lesions, the proximity of important structures which cannot be sacrificed, and the extreme technical difficulties of the procedure. Whether or not operation shall be done must be decided on the merits of the individual circumstances, but it is well to remember, before deciding against it in any case, that, as Kocher once put it, "the worst which happens to such patients may not be inconsistent with the alleviation of human suffering."

For anatomic reasons, as we have just implied, operation in this area is always technically very difficult, and, while the highly fatal character of the growth warrants and its highly malignant character demands radical surgery, the physiologic result of the necessary anatomic mutilation cannot be ignored or lightly dismissed. One must do more than save the patient's life; one must save him for a life that is worth living. A brief consideration of the regional anatomy and the physiologic function of the structures in question will make that point clear.

In man the main pancreatic duct, or duct of Wirsung, enters the duodenum at the ampulla of Vater, usually after fusion with the common duct, less often by an independent, parallel channel. Occasionally in

humans and always in animals an accessory pancreatic duct, the duct of Santorini, enters the duodenum about an inch above the ampulla of Vater. Through these ducts are discharged into the intestinal canal the secretions which are essential for certain phases of digestion.

The pancreatic secretion is essential for the digestion of all proteins and for the digestion of some fats. The gastric pepsin reduces proteins to acid albumins, polypeptides, tripeptides, and dipeptides; but a pancreatic secretion, trypsin, is necessary to break them down into the ultimate end product, amino acids, and another pancreatic secretion, nuclease, is necessary to break down nucleoproteins or compound proteins into more simple protein radicals. The gastric lipase, by a process of emulsification, converts some fats into fatty acids and glycerol, but other fats pass into the duodenum in a neutral form and the joint action of the intestinal and pancreatic lipase is necessary to convert them into fatty acids and glycerol. In addition, though we shall not concern ourselves with it in this communication, the action of bile is necessary for other processes of digestion.

From even this very elementary statement of anatomic and physiologic facts it will be clear that the most conservative radical surgery in this region, if we may use such a paradox, will always result in unintentional or deliberate mutilation of the common and pancreatic ducts and will frequently result in damage to the head of the pancreas also. From this anatomic mutilation will arise another result, an alteration of the metabolic processes involving the digestion of proteins and fats.

The technical difficulties of surgery in the ampullar region and the physiologic disturbances associated with the anatomic alterations probably explain, as Outerbridge suggested, why for so many years malignancy in this area was considered beyond surgical attack. In the 110 cases of ampullar cancer which he collected in 1913, only 20 had been handled by radical surgery, and twenty-two years later, when Hunt and Budd[6] again reviewed the literature, they accepted only 76 surgical cases as authentic, including 3 which they reported themselves. Only a few cases have been added since (by Whipple, Parsons, and Mullins,[7] Cooper,[3] Hoffman and Pack,[8] and Stewart and Lieber[9]), and the total number of recorded cases, after the elimination of duplicates, probably does not exceed a hundred.

In 20 of the 76 cases collected by Hunt and Budd transduodenal resection of the tumor was done with 9 deaths, 45 per cent. In 19 cases the same procedure was done in combination with reimplantation of the pancreatic duct or of the common duct or of both with 4 deaths, 21 per cent. In 13 cases internal or external drainage was added to the combined procedure with 6 deaths, 46 per cent. In other words, in 63 of the 76 cases transduodenal resection of the tumor was done either alone or in combination with other procedures with a mortality of 24,

38 per cent. In the remaining 13 cases resection of the duodenum was done 9 times with 3 deaths, 33 per cent, and retroduodenal excision of the tumor in 4 cases with 2 deaths, 50 per cent.

Thirty-eight per cent is a very discouraging surgical mortality, even if it be remembered that every patient who died after operation would have died eventually, and probably promptly, from the effects of his disease. Equally discouraging is Cooper's estimate that there were probably not more than 7 permanent cures, 9 per cent, in the whole series, though again we must remember that without surgery there would have been no cures at all. It is only fair to state, too, that at least 1 patient lived as long as twenty-two years after operation. Another hopeful feature of the situation is pointed out by Hunt and Budd: in the 43 cases collected by Cohen and Colp[10] in 1927, the mortality was 43 per cent, but in the 18 cases which they themselves added to the compilation the mortality was only 22 per cent.

But even that reduced mortality is still far too high, and many authors feel that it might be further decreased if certain risks could be eliminated, such as are associated with implantation of the head of the pancreas into the small bowel or reimplantation of the pancreatic duct. Such procedures require tedious dissection; they prolong the operation; they increase the likelihood of shock; and peritonitis is a rather frequent and highly fatal complication, chiefly because of the absence of protective peritoneal covering in parts of this area and because of the highly potent character of the pancreatic secretion. Before the continuity of the pancreas and the intestinal tract can be destroyed, however, two points must be clearly established. In the first place, it must be demonstrated that the deprivation of the pancreatic secretion has no significant effect upon the digestion of fat and protein. In the second place, it must be demonstrated that the life expectancy is not altered by its withdrawal from the intestinal tract. The various investigators who have already worked on this problem have apparently answered those questions to their own satisfaction. We have approached it from a somewhat different angle and we doubt the soundness of all their conclusions.

With the idea of studying the effect on protein and fat digestion and assimilation of various procedures designed to deprive the intestinal tract of the external secretion of the pancreas, we performed on a series of experimental animals complete pancreatectomy, partial pancreatectomy, partial pancreatectomy with ligation and division of the pancreatic ducts, and simple ligation and division of the pancreatic ducts. The two protocols which we quote below illustrate the widely different effect upon the liver when the pancreas is left in situ and when it is partially excised.

Dog 20A.—Ligation and division of the pancreatic ducts was done under ether anesthesia Dec. 30, 1935, and the animal was sacrificed Aug. 11, 1937. It reacted promptly from the operation and was in excellent health and nutrition throughout

the period of observation. At no time were there any but trivial and transient alterations in the blood chemistry. As will be noted from Table I, digestive studies were made after the administration of a special diet, which will be commented upon later.

At autopsy the pancreas, which was small and firm, was fibrous on section. The common duct was patent throughout its course, but the pancreatic ducts, which had been ligated and divided at their point of exit from the pancreas, were impervious to the passage of a probe, and it could be clearly demonstrated that no pancreatic secretion could have entered the duodenum. The other organs were grossly normal. The microscopic findings follow:

Liver.—The arrangement of the liver cells and the liver cell cords was orderly. The cell cords in the central two-thirds of the lobules were slightly narrower than normal and the cytoplasm of the cells contained occasional minute rusty-brown

TABLE I

FAT AND PROTEIN DIGESTION FOLLOWING VARIOUS FORMS OF PANCREATIC EXCLUSION

		FAT			PROTEIN		
		IN DIET	IN STOOLS	% DIGESTED	IN DIET	IN STOOLS	% DIGESTED
Approximately 8 Weeks After Operation							
Dog 9A 8.63 kg. Mixture*	(a)	163.62 gm.	8.76 gm.	94.65	106.83 gm.	9.63 gm.	91.0
	(b)	79.30 gm.	1.16 gm.	98.53	83.48 gm.	1.71 gm.	97.95
Dog 15A 4.54 kg. Mixture	(a)	27.68 gm.			18.74 gm.		
Dog 17A 6.59 kg. Mixture	(a)	124.86 gm.	4.4 gm.	96.5	81.54 gm.	6.63 gm.	92.0
	(b)	60.84 gm.	0.51 gm.	99.16	64.04 gm.	1.14 gm.	98.2
Dog. 19A 6.93 kg. Mixture	(a)	131.28 gm.	3.15 gm.	97.6	85.74 gm.	5.42 gm.	93.7
	(b)	31.94 gm.			33.62 gm.		
Dog 20A 7.84 kg. Mixture	(a)	148.23 gm.	5.35 gm.	96.4	96.99 gm.	15.1 gm.	84.5
	(b)	72.28 gm.	3.91 gm.	94.6	78.16 gm.	6.34 gm.	91.9
Approximately 20 Months After Operation							
Dog 9A 9.09 kg. Mixture	(a)	104 gm.	4.2 gm.	96.0	64.9 gm.	4.4 gm.	93.3
		114.8 gm.	3.6 gm.	96.9	74.98 gm.	6.1 gm.	91.9
	(c)	146.4 gm.	14.6 gm.	90.0	73.4 gm.	2.8 gm.	96.2
		135.7 gm.	12.3 gm.	89.3	62.5 gm.	2.0 gm.	95.5
Dog 20A 12.27 kg. Mixture	(a)	154.96 gm.	6.2 gm.	96.0	101.2 gm.	4.3 gm.	95.7
	(a)		4.6 gm.	97.0		4.6 gm.	95.4
	(c)	198.2	25.0 gm.	87.4	99.2 gm.	0.5 gm.	99.15
	(c)		15.9 gm.	92		4.1 gm.	95.9

*Proportions of carbohydrate, fat, and protein in diet. Amount given on basis of 80 calories per day per kg. of body weight. Corrections made for uneaten quantities. (a) 51.24 gm. CHO, 30.07 gm. fat, 19.64 gm. protein—79.8 calories per kg. body weight. (b) 60.02 gm. CHO, 19.78 gm. fat, 20.82 gm. protein—80.05 calories per kg. body weight. (c) 40.26 gm. CHO, 39.92 gm. fat, 20.30 gm. protein—79.99 calories per kg. body weight.

granules. The central vein and the adjacent capillaries were slightly distended. The structures of the periportal space were well preserved and no cellular infiltration was seen. The cells lining the larger bile ducts were well preserved; they were of the tall columnar type and were thrown into coarse folds in some areas.

Spleen.—The septa and capsules were of normal width. The malpighian corpuscles were large, and few and far apart; they exhibited active germinal centers. The sinusoids of the pulp were either empty or filled with red blood cells. The surrounding stroma seemed slightly increased.

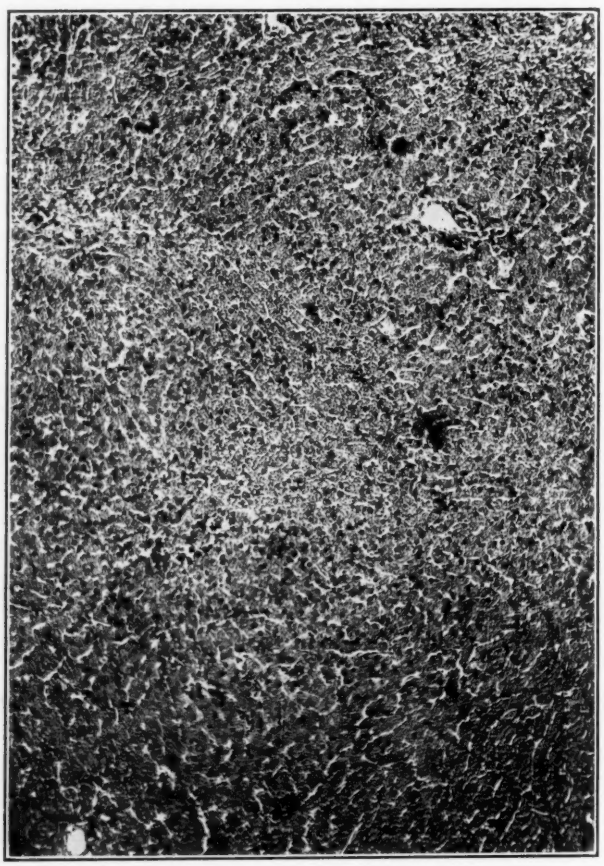

Fig. 1.—Showing liver of apparently normal architecture after ligation and division of both pancreatic ducts. See protocol of Dog 20A for details.

Thyroid.—The acini, which varied in size, were lined by low cuboidal cells and were filled with colloid. Groups of interacinar cells were frequent. The acini were arranged in lobules of varying size and were separated by delicate connective tissue septa.

Kidneys.—The glomeruli were of normal size and orderly arrangement. In many areas the space between Bowman's capsule and the capillary was widened. The lumina of the convoluted tubules were open and the lining cells, which were well preserved, exhibited vacuolated cytoplasm. The interstitial connective tissue and the blood vessels showed no changes. The urothelium lining the calix and the renal pelvis was well preserved.

Almost similar post-mortem findings were noted in Dog 2A, which was submitted to total pancreatectomy and which lived for five days; during this period insulin was administered at intervals and lecithin was added to the diet. Similar findings were also noted in Dog 17A, which was submitted to partial pancreatectomy and ligation and division of the pancreatic ducts. This dog lived for 100 days, during which time lecithin was added to the diet at intervals and he was submitted to the special studies shown in Table I.

Dog 9A.—Partial pancreatectomy and ligation and division of both pancreatic ducts was performed under ether anesthesia Dec. 30, 1935, and the animal was sacrificed Aug. 11, 1937. This dog was submitted to the same studies as Dog 20A, as shown in Table I. It recovered promptly from the operative procedure, remained in good health throughout the period of observation, and was in excellent condition at death. There were no significant changes in the blood chemistry at any time. At post mortem the remnant of the pancreas, which was found only after tedious dissection, was very small and very hard. The spleen was somewhat smaller than normal and grossly exhibited a mild degree of fibrous change. There were no other positive findings.

The microscopic findings follow:

Liver.—The normal lobular arrangement of the liver cells and liver cell cords was scarcely discernible, and with but few exceptions the cells, both in the center and the periphery of the lobule, were transformed into large vacuoles with peripherally placed nuclei. The "signet ring" appearance of the liver cells was marked. The structures in the periportal area were intact, but some of the central veins and adjacent capillaries were distended with blood.

Pancreas.—The cells were arranged in columns and sheaths rather than according to the arrangement of normal acini. They were polyhedral, the cytoplasm stained lightly, and the nuclei were large and vesicular. In some areas delicate connective tissue septa divided large cell groups into lobules. Here and there small groups of lighter-staining cells were seen, which resembled islet cells of Langerhans. In the intralobular connective tissue were seen ducts whose lumina were lined by cylindrical cells.

Spleen.—The capsule was delicate. The septa were of normal width. Malpighian corpuscles were numerous and of normal size, with active germinal centers. Scattered throughout the pulp were black and rusty-brown granules, and the connective tissue element was somewhat increased. All the findings were suggestive of passive congestion.

Thyroid.—The acini varied in size, but all contained light, pink-staining colloids and were lined by flat cells. Groups of interacinar cells were frequent. The acini were grouped into lobules separated by scanty fibrous connective tissue stroma. There were no areas of hemorrhage or necrosis. The gland showed in certain areas a slight activity, but nowhere was there any evidence of atrophy.

Kidneys.—The glomeruli were of normal size and orderly arrangement. In a few areas the space between Bowman's capsule and the capillary was widened. The lumina of the convoluted tubules were open and the lining cells, which were quite well preserved, exhibited vacuolated cytoplasm. The stroma and blood vessels showed no changes. There was an occasional accumulation of small round cells in the tunica propria of the calyx.

Almost similar findings were noted in the dog submitted to total pancreatectomy and kept alive five days by the use of insulin (Dog 1A) and in the dog submitted to partial pancreatectomy and sacrified eighty days later, after a series of studies in fat and protein as shown in Table I (Dog 15A). Another dog (19A) was submitted to ligation and division of the pancreatic ducts and was studied with a special diet as shown in Table I. The animal was in excellent condition during the whole

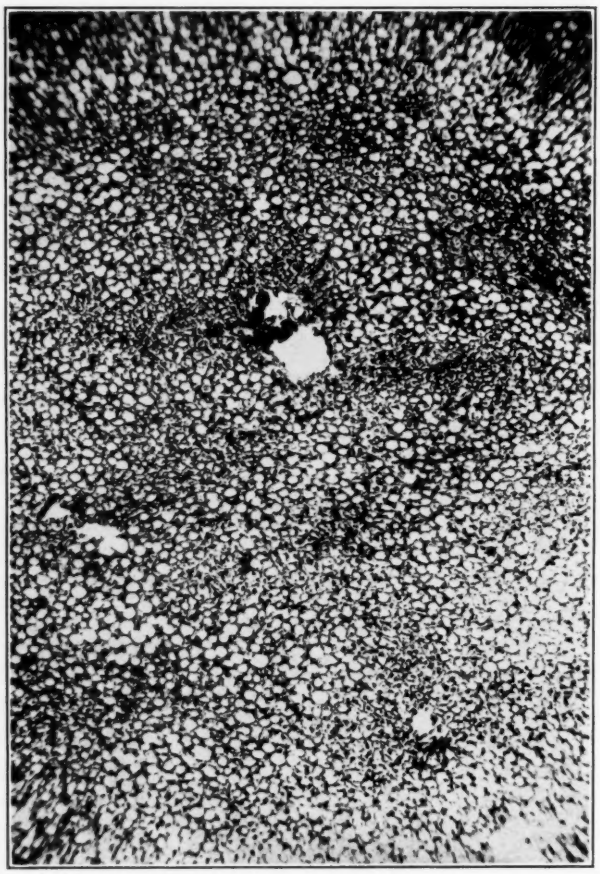

Fig. 2.—Showing liver with marked vacuolization of liver cells, following partial pancreatectomy with ligation and division of both pancreatic ducts. See protocol of Dog 9A for details.

period of observation, but post-mortem studies are unfortunately not available as he was stolen from his cage thirteen months after operation.

Although the intestinal tract was deprived of the external secretion of the pancreas by the various procedures which we have listed, neither the protein nor the fat digestion was apparently affected, as Table I shows. By the special diet which we employed, consisting of cracker meal, lard, and lean meat, it was possible to determine with approximate accuracy the amount of carbohydrate, fat, and protein ingested and to correlate the intake with the fecal output. The diet was administered

for several days before the stools were studied. The feedings were carefully weighed, and uneaten portions were also weighed and subtracted from the total amount given. Powdered charcoal in capsules was put into the stomach before the feedings were begun, and capsules containing carmine were introduced at the end of the test. The stools were thus so clearly marked that it was possible to determine exactly which feces were the excreted product of the exact amount of food ingested. The studies made approximately eight weeks after operation, as well as the studies made almost twenty months later (Table I), clearly demonstrate that, in spite of the exclusion of the external secretion of the pancreas from the intestinal tract, the animals were able to digest an unusually high fat and protein diet. Similar clinical studies have been made in the successful case reported by Whipple, Parsons, and Mullins, in which their own newly devised operation was used.

On the other hand, after the intestinal tract has been deprived of pancreatic secretion, the liver has an apparently abnormal capacity to assimilate fat. This observation was first made by von Mering in 1889 and by Naunyn in 1898 (quoted by Aylward and Holt[11]) and has been corroborated by other workers in this field. Such changes, as Hershey, Best, and others have shown (Aylward and Holt), can be mitigated or prevented altogether by the use of lecithin or choline or by the use of raw pancreas. Our own experiments clearly show that by the use of lecithin the animals deprived of the external secretion of the pancreas could tolerate an unusually high fat and protein diet without developing the fatty changes in the liver which, in an extreme degree, are not compatible with life.

It will be noted that in Dog 2A, which was submitted to complete pancreatectomy, and in Dog 17A, which was submitted to excision of the head and body of the pancreas plus ligation and division of the pancreatic ducts, the pancreatic secretion not only was excluded from the intestinal tract but also was practically eliminated from the body except on the assumption that both animals had aberrant pancreatic tissue in abnormal locations, which would be a rather unlikely coincidence. Both of these dogs were fed lecithin and neither developed the fatty changes in the liver which we have just described.

But neither did such changes develop in Dog 20A, in which an entirely different set of circumstances was present. In this experiment the pancreatic secretion was excluded from the intestinal tract, but functioning pancreatic tissue was left in situ. The animal lived for twenty months, was in excellent health and nutrition when sacrificed, and at autopsy revealed no evidence of fatty metamorphosis of the liver. Neither lecithin nor any other agent was given to forestall the changes.

In view of these findings it seems reasonable to assume the existence of some internal secretion of the pancreas which affects the metabolism of the liver cell and which, when the pancreas itself is left undisturbed,

will prevent fatty metamorphosis in the liver even after the external pancreatic secretion has been excluded from the intestinal tract by ligation and division of the pancreatic ducts. This reasoning is along the lines of Dragstedt's[12, 13] assumption that the beneficial effects of the administration of raw pancreas after complete pancreatectomy are due to some specific substance in the pancreas, which he has termed a fat-metabolizing hormone and which he has named lipocaic.

The experimental work which we have just outlined has a clear clinical application. It would seem to prove that, from the standpoint of the digestion of protein and fat, the radical surgery necessary for the cure of tumors of the ampullar and periampullar region is entirely feasible. But it would also seem to prove that some qualifications or reservations are necessary from the standpoint of the metabolism of the liver cells whenever such surgery involves excision of any part of the pancreas.

When the pancreas is not involved in the malignant process and can be left undisturbed, radical surgery may be safely done and the pancreatic duct ligated and divided but not reimplanted. Even though the external secretion of the pancreas is thus excluded from the intestinal tract, fat and protein digestion will continue normally, or within approximately normal limits, and the normal metabolism of the liver cells will be unaffected because there will be no disturbance of the hypothetical internal secretion of the pancreas responsible for that metabolic function.

On the other hand, when the pancreatic tissue is involved in the malignant process and even partial pancreatectomy is a necessary part of the operation, then the circumstances are very different. The digestion of fat and protein by way of the intestinal canal will continue normally or within normal limits, but fatty metamorphosis of the liver will occur because there has been a disturbance of the hypothetical internal secretion of the pancreas responsible for the normal metabolism of the liver cell. Unless some provision is made to counteract the resulting abnormal fatty metamorphosis of liver cells, the life expectancy will be materially reduced and the burden of any intercurrent disease, especially of any infectious disease, might well prove intolerable. Fortunately such a safeguard would seem to be found in lecithin, or choline, or in the alcoholic extract of the pancreas demonstrated by Dragstedt.

The brilliant results reported by Whipple, Parsons, and Mullins in the case in which their own operation was used and the case of subtotal pancreatectomy in a child not quite 2 years old which was reported by Graham[14] might seem to invalidate these contentions. The operation devised by Whipple and his associates is a two-stage procedure which, from the standpoint of this discussion, involves a V-shaped excision of the head of the pancreas as well as ligation and division of the pancreatic ducts. In Graham's case, in which the indication was an intractable

hypoglycemia, only a small strip of pancreas was left to protect the common duct. Both patients were in good condition for some time after operation. Graham, discussing the contribution of Whipple and his associates, reported the present condition of his patient, which was still good at the end of more than one year, and made the point that if a rapidly growing child could get along without the external secretion of the pancreas, certainly an adult should be able to do so also.

Without detracting in any way from the immediate brilliant results of these procedures, it seems justifiable to question whether they can be expected to continue without the use of some agent to forestall liver damage. Studies in fat and protein digestion do not seem to be enough to settle the question. In our own experimental animals fatty changes in the liver developed in spite of normal digestion of these substances in the intestinal tract in all the animals which were not fed lecithin to forestall them. Is it not possible that similar changes will occur clinically under the same circumstances?

We shall not discuss the question of two-stage surgery for tumors of the ampullar region except in a very general way. The plan was first suggested by W. J. Mayo (quoted by Hunt and Budd) and is a part of the procedure advocated by Whipple and his associates. The formation of an external biliary fistula to correct the cholemia is efficacious, but it is obviously both dangerous and objectionable, and some form of internal drainage, preferably by way of the stomach, is decidedly better. Such a method provides for prolonged drainage of the biliary system without the loss of bile, and, if the patient survives, puts him into better condition to withstand later radical extirpation. Of course, he does not always survive. Hunt and Budd make the point that, while the mortality for stage surgery in their collected series is apparently low, 2 deaths in 8 cases, it is by no means as low as it seems, for it makes no allowance for the many patients subjected to the first operation who do not live to withstand the second. It must be remembered that in such patients the mere opening of the abdomen is attended with a high mortality. Clute's[15] figures for carcinoma of the pancreas show that simple exploration reduces the life expectancy from 7.2 months in the untreated case to 3 months in the explored case.

Jaundiced patients, as we have already mentioned, are inherently poor surgical risks and must never be operated on without careful study and adequate preparation. We[16] personally have found in a large series of cases that the Quick hippuric acid liver function test is an adequate method for determining the initial hepatic function and for checking its improvement under preparation. We have also found that the serum volume test[17] for the hemorrhagic diathesis in jaundice, which we have personally devised, has yielded very satisfactory results in demonstrating a possible hemorrhagic tendency and in checking its improvement under appropriate therapy. It seems scarcely necessary to

emphasize that the liberal use of glucose in various forms, especially its oral administration, is the basis of the preoperative preparation. Transfusion should be employed as indicated, and oxygen, as suggested by Judd, is also helpful.

Except in the liver and in the remnant of pancreas left in situ, we found no significant changes at autopsy in any of our experimental animals. The kidneys were normal and the spleens showed only mild fibrotic changes. Sweet[18] mentions an atrophy of the thyroid gland in pancreatectomized dogs, but Whipple and his coworkers did not note this change in their cases which came to autopsy, nor did we find it. On the contrary, our experimental animals exhibited a slight activation, though not enough to be called pathologic. We found no ulceration in the gastrointestinal tract in any of the animals which we observed.

We plan, as the next step in this investigation, to study the effects of total excision of the duodenum in combination with exclusion of the pancreatic secretion by various methods, particularly as regards the digestion of fat and protein in the intestinal tract and the development of fatty changes in the liver.

SUMMARY

1. The outstanding considerations in various types of cancer of the ampullar and periampullar regions are outlined, with particular reference to the physiologic disturbances which follow the necessary radical surgery for the cure of these tumors.

2. The types of operation devised for these lesions are considered from the standpoint of the immediate surgical mortality.

3. Personal experiments are outlined, in which the external pancreatic secretion was excluded from the intestinal tract by various methods. (a) In all the animals the digestion of fat and protein, even when a diet unusually rich in these substances was given, was approximately normal. (b) Fatty changes in the liver developed in spite of this fact in the animals in which partial or complete pancreatectomy was done; (c) but such changes did not develop in animals in which partial or complete pancreatectomy was done when lecithin was added to the diet. (d) Nor did they develop in animals in which the pancreatic ducts were ligated and divided but the pancreas was left in situ.

4. The experimental evidence outlined above would seem to corroborate Dragstedt's assumption of a pancreatic hormone which is able to take over the function hitherto assigned exclusively to the external secretion.

5. The clinical application of this experimental work suggests: (a) that radical surgery which does not involve excision of the pancreatic tissue may safely be done without reimplantation of the pancreatic ducts for malignancies of the ampullar and periampullar regions; in such

cases fatty infiltration of the liver is not likely to develop; (b) that when such surgery must include excision of the head or of the head and body of the pancreas, fatty changes will develop within the liver unless lecithin, choline, or pancreatic substance or alcoholic extracts of pancreas be administered to forestall them.

6. Stage surgery for malignancies of this area is briefly considered, together with the general principles of preoperative preparation.

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THE EFFECT OF EPHEDRINE ON PANCREATIC SECRETION: A METHOD FOR THE MANAGEMENT OF PATIENTS HAVING A PANCREATIC FISTULA*

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INTRODUCTION

THE case of a patient with a fistula from which activated pancreatic juice flows frequently constitutes a serious problem. This is because the skin and tissues with which the juice comes into contact are markedly irritated and digested and occasionally large deep ulcers result. This is especially true when large quantities of the digestive fluid flow from the fistula. Various means have been proposed to reduce the volume of juice secreted. A careful review of the literature, however, showed, as is quite well known among surgeons, that none of the methods proposed have, in general, proved to be successful. Accordingly this investigation has been undertaken with the idea in mind (a) of finding some relatively innocuous method of reducing the volume output of pancreatic juice without fasting the subject and (b) of testing the suitability of silica gel as a dressing powder. Further, in patients having a pancreatic fistula which drains fluid free of active trypsin, it is logical to assume that any procedure which would reduce the volume of secretion would promote closure of the fistula with granulation tissue, since most such fistulas tend to close spontaneously sooner or later.

REVIEW OF THE LITERATURE

The chief method which has been employed clinically to reduce pancreatic secretion is that proposed by Wohlgemuth.[1] This method consists in feeding a low carbohydrate, high fat and protein diet (an "antidiabetic diet") along with the administration of sodium bicarbonate. The method was based on the observations of Pavlov[2] and Wohlgemuth,[1] who reported that carbohydrate food caused the production of larger quantities of pancreatic juice than proteins (less) and fats (least). They also reported that sodium bicarbonate given orally decreased pancreatic secretion.

The Wohlgemuth measure has been used in a number of patients with pancreatic fistula. A review of the literature by Desvaux de Lyf[3] in 1912 records that the measure had been used in 17 cases. In 10 the measure apparently decreased the secretion; in 7 it did not. Since then reports have been rather evenly divided between favorable[4-9] and unfavorable[10-14] results.

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The variable results reported may be explained either by assuming that the Wohlgemuth measure had no effect on the course of the tissue changes leading to a closure of the fistula, or, when the flow of secretion appeared to be decreased by the diet and alkali, the decrease was within the normal variations in the output of juice via the fistula. In regard to the inhibitory effect of alkali on pancreatic secretion, the literature is not consistent. Although most observers report a decrease in secretion after sodium bicarbonate,[1, 2, 15, 30] Glaessner[16] observed that sodium bicarbonate was without effect in a patient in whom the duct of Wirsung was directly cannulated, and W. N. Boldyreff and E. B. Boldyreff[17] and W. N. Worobjeff[18] record that only concentrated solutions of alkali inhibit pancreatic secretion in the dog.

In regard to the response of the pancreas to various foodstuffs, the experimental data on man and dog indicate that bread or carbohydrates stimulate pancreatic secretion most, proteins next, and fats least. However, that data published are not adequate to rule out the factor of spontaneous variation from day to day. Yet, in my opinion the literature indicates that pancreatic secretion tends to be decreased by the administration of sodium bicarbonate and a diet high in fat, moderate in protein, and low in carbohydrate.

In regard to the use of drugs atropine has been employed in managing patients with pancreatic fistula. The results have been variable. It is well known that atropine has no effect on the chemical or secretin phase of pancreatic secretion which is the most important phase. Atropine, however, does abolish the nervous (vagus) phase of pancreatic secretion.[19, 20] This drug also should decrease the pancreatic response to a meal, because it depresses the output of gastric juice, the acid of which causes secretin production.

The only drug or procedure which is known to decrease the response of the pancreas to secretin, without causing harmful side reactions, is epinephrine. This was first shown by Edmunds.[21] Mann and McLachlin[22] observed inhibition of pancreatic secretion caused by secretin on the injection of a dose of epinephrine (1:500,000) which causes a fall in blood pressure, which, however, caused a decrease in the volume of the gland, showing that the blood vessels of the pancreas are especially susceptible to the vasoconstrictor action of epinephrine. Thus the pancreas is very susceptible to changes in the blood flow, a fact demonstrated by Babkin.[23] Barlow[24] has verified the importance of the relation of blood flow to the secretory response of the pancreas by showing that vasodilations act synergistically with secretin.

The effect of ephedrine, a drug which has a pharmacologic action somewhat like that of epinephrine, on the response of the pancreas to secretin has not been studied. Because ephedrine causes a general vasoconstriction and because it has a more prolonged action than epinephrine, it was thought that ephedrine should reduce the pancreatic

secretory response to a meal. Accordingly experiments were devised to determine the effect of ephedrine on pancreatic secretion.

METHOD OF PRODUCTION OF A TOTAL PANCREATODUODENOFISTULA

This procedure involves two operations; the first stage consists of the production of an intestinal fistula through which the pancreatic juice might be returned to the animal. About 6 inches of the jejunum is resected, and the proximal end is brought to the outside through a stab wound; then an end-to-side anastomosis is done between the distal end of the resected bowel and the jejunum, after an end-to-end anastomosis had been performed between the two cut ends of the jejunum. Three or four weeks later the second stage is carried out. Under ether or nembutal anesthesia, the abdomen is opened, the duodenum freed and brought into the abdominal opening. The accessory duct (Santorini) is ligated

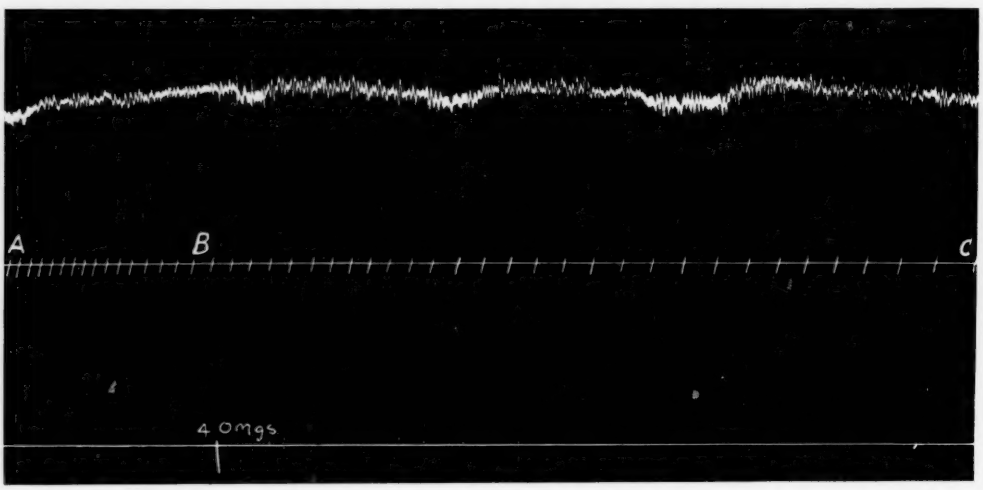

Fig. 1.—Top line: blood pressure curve. Middle line: *A-B* shows control number of drops of pancreatic juice secreted in response to the continuous injection of secretin intravenously at the rate of 2.75 c.c. per minute; such a rate of injection of the secretin was maintained throughout this experiment. *B-C* shows decrease in number of drops of pancreatic juice after the injection of 4 mg. of ephedrine sulfate subcutaneously. Lower line: Time of injection of 4 mg. of ephedrine sulfate subcutaneously.

and divided, as are any other accessory ducts should they be present. Then the duodenum is divided just below the entrance of the common bile duct, and again about 1 inch below the entrance of the main duct (Wirsung) of the pancreas. An end-to-end anastomosis is done between the cut ends of the duodenum. The upper end of the resected portion of the duodenum is turned in and closed tightly, as is the lower end after the insertion into the lumen of a suitable piece of tubing, attached to a glass collar. The rubber tubing is brought out through a stab wound and connected to a rubber balloon.

Postoperative Care of the Animals.—After each operation, the animals were hyperventilated with 5 per cent CO_2 and 95 per cent O^2. Daily subcutaneous injections of 500 to 800 c.c. of normal saline solution were

given throughout the experiment. Food was withheld until the third day, at which time 300 c.c. of milk and a small amount (¼ pound) of dog food, liver, or hamburger is fed to the animal. The diet was increased from day to day until 500 c.c. of milk and 1 pound of dog food constituted the animal's rations. The pancreatic secretion was collected every 4 hours during the first 48 hours and then every hour daily from 8:00 A.M. until 8:00 P.M., and as one collection from 8:00 P.M. until 8:00 A.M. The secretions were collected under sterile precautions and were returned via the intestinal fistula to the small intestine. After suitable control periods were obtained for the daily hourly collections, 10 mg. of ephedrine were injected subcutaneously every 2 hours on the test days.

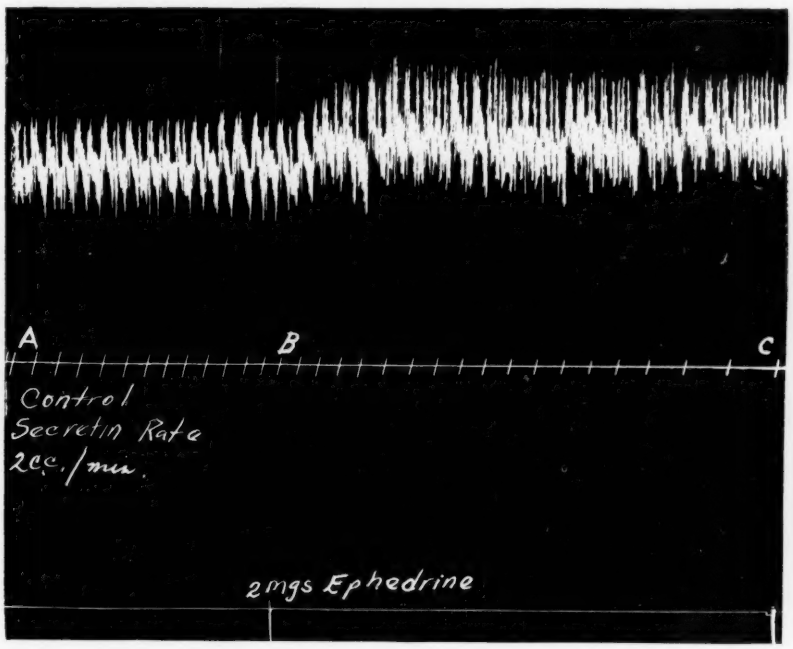

Fig. 2.—Top line: Blood pressure curve. Middle line: *A-B* shows control number of drops of pancreatic juice secreted in response to the continuous injection of secretin intravenously at the rate of 2 c.c. per minute; such a rate of injection of the secretin was maintained throughout this experiment. *B-C* shows decrease in number of drops of pancreatic juice after the injection of 2 mg. of ephedrine sulfate subcutaneously. Lower line: Time of injection of 2 mg. of ephedrine sulfate subcutaneously.

After each test day, the hourly secretion was allowed to return to the level of the previous control day before the treatment with ephedrine was repeated.

RESULTS

Four acute experiments were performed. The accessory pancreatic duct was ligated and the main duct cannulated, and, by means of an electric drop counter, each drop of pancreatic secretion was recorded on a very slowly revolving drum. The secretion of pancreatic juice was kept at a more or less constant level throughout the experiments by the

TABLE I

RESULTS OBTAINED AFTER THE SUBCUTANEOUS INJECTION OF 10 MG. OF EPHEDRINE EVERY 2 HOURS WITH CONTROL DAYS BEFORE AND AFTER TREATMENT

HOUR	I CONTROL (DAY)	I TREATMENT (DAY)	I CONTROL (DAY)	II CONTROL (DAY)	II TREATMENT (DAY)	II CONTROL (DAY)	III CONTROL (DAY)	III TREATMENT (DAY)	III CONTROL (DAY)
	Basal =	Basal =	Basal =	Basal =	Basal =	Basal =	Basal =	Basal =	Basal =
8-9 A.M.	30 c.c.	28 c.c.	35 c.c.	26 c.c.	30 c.c.	29 c.c.	29 c.c.	20 c.c.	25 c.c.
10 A.M.	27 c.c.	17 c.c.	38 c.c.	25 c.c.	7 c.c.	26 c.c.	26 c.c.	11 c.c.	25 c.c.
11 A.M.	29 c.c.	20 c.c.	38 c.c.	20 c.c.	21 c.c.	30 c.c.	30 c.c.	9 c.c.	22 c.c.
12 M.	32 c.c.	11 c.c.	35 c.c.	24 c.c.	9 c.c.	24 c.c.	24 c.c.	14 c.c.	29 c.c.
1 P.M.	42 c.c.	40 c.c.	38 c.c.	22 c.c.	29 c.c.	25 c.c.	25 c.c.	12 c.c.	26 c.c.
2 P.M.	51 c.c.	10 c.c.	35 c.c.	21 c.c.	16 c.c.	19 c.c.	19 c.c.	23 c.c.	27 c.c.
3 P.M.	39 c.c.	44 c.c.	26 c.c.	18 c.c.	13 c.c.	17 c.c.	17 c.c.	23 c.c.	25 c.c.
4 P.M.	35 c.c.	25 c.c.	28 c.c.	22 c.c.	21 c.c.	21 c.c.	21 c.c.	15 c.c.	22 c.c.
5 P.M.	31 c.c.	48 c.c.	29 c.c.	26 c.c.	16 c.c.	19 c.c.	19 c.c.	10 c.c.	22 c.c.
6 P.M.	29 c.c.	36 c.c.	29 c.c.	26 c.c.	15 c.c.	30 c.c.	30 c.c.	11 c.c.	22 c.c.
7 P.M.	10 c.c.	29 c.c.	28 c.c.	21 c.c.	20 c.c.	26 c.c.	26 c.c.	10 c.c.	28 c.c.
8 P.M.	45 c.c.	29 c.c.	30 c.c.	20 c.c.	17 c.c.	22 c.c.	22 c.c.	13 c.c.	23 c.c.
Total	400 c.c.	337 c.c.	389 c.c.	290 c.c.	214 c.c.	288 c.c.	288 c.c.	148 c.c.	296 c.c.
	Per cent reduction = 15.75 per cent			Per cent reduction = 26.2 per cent			Per cent reduction = 47.91 per cent		

HOUR	IV CONTROL (DAY)	IV TREATMENT (DAY)	IV CONTROL (DAY)	V CONTROL (DAY)	V TREATMENT (DAY)	VI CONTROL (DAY)	VI TREATMENT (DAY)
	Basal =	Basal =	Basal =	Basal =	Basal =	Basal =	Basal =
8-9 A.M.	16 c.c.	14 c.c.	18 c.c.	15 c.c.	18 c.c.	15 c.c.	18 c.c.
10 A.M.	17 c.c.	9 c.c.	11 c.c.	11 c.c.	15 c.c.	19 c.c.	11 c.c.
11 A.M.	14 c.c.	12 c.c.	19 c.c.	19 c.c.	6 c.c.	18 c.c.	16 c.c.
12 M.	16 c.c.	8 c.c.	19 c.c.	19 c.c.	17 c.c.	17 c.c.	10 c.c.
1 P.M.	16 c.c.	15 c.c.	20 c.c.	20 c.c.	7 c.c.	27 c.c.	19 c.c.
2 P.M.	12 c.c.	7 c.c.	19 c.c.	19 c.c.	14 c.c.	20 c.c.	11 c.c.
3 P.M.	14 c.c.	14 c.c.	21 c.c.	21 c.c.	9 c.c.	22 c.c.	6 c.c.
4 P.M.	14 c.c.	10 c.c.	18 c.c.	18 c.c.	17 c.c.	19 c.c.	11 c.c.
5 P.M.	17 c.c.	19 c.c.	19 c.c.	19 c.c.	9 c.c.	15 c.c.	22 c.c.
6 P.M.	20 c.c.	9 c.c.	17 c.c.	17 c.c.	16 c.c.	18 c.c.	9 c.c.
7 P.M.	18 c.c.	10 c.c.	16 c.c.	16 c.c.	7 c.c.	18 c.c.	15 c.c.
8 P.M.	15 c.c.	10 c.c.	19 c.c.	19 c.c.	12 c.c.	20 c.c.	7 c.c.
Total	189 c.c.	137 c.c.	203 c.c.	203 c.c.	145 c.c.	218 c.c.	145 c.c.
	Per cent reduction = 27.51 per cent			Per cent reduction = 25.61 per cent		Per cent reduction = 33.48 per cent	

TABLE I—CONT'D

HOUR	VII CONTROL (DAY)	VII TREATMENT (DAY)
	Basal =	Basal =
8-9 A.M.	11 c.c.	13 c.c.
10 A.M.	12 c.c.	7 c.c.
11 A.M.	9 c.c.	9 c.c.
12 M.	13 c.c.	6 c.c.
1 P.M.	12 c.c.	11 c.c.
2 P.M.	12 c.c.	9 c.c.
3 P.M.	10 c.c.	15 c.c.
4 P.M.	11 c.c.	8 c.c.
5 P.M.	10 c.c.	9 c.c.
6 P.M.	10 c.c.	8 c.c.
7 P.M.	12 c.c.	10 c.c.
8 P.M.	13 c.c.	6 c.c.
Total	135 c.c.	111 c.c.
Per cent reduction = 17.85 per cent		

HOUR	VIII CONTROL (DAY)	VIII TREATMENT (DAY)	VIII EPHEDRINE	VIII CONTROL (DAY)
	Basal =	Basal =		Basal =
8-9 A.M.	29 c.c.	62 c.c.	10 mg.	30 c.c.
10 A.M.	26 c.c.	46 c.c.		27 c.c.
11 A.M.	42 c.c.	47 c.c.	10 mg.	29 c.c.
12 M.	22 c.c.	21 c.c.		32 c.c.
1 P.M.	43 c.c.	35 c.c.		42 c.c.
2 P.M.	52 c.c.	52 c.c.	10 mg.	51 c.c.
2:30-3:30 P.M.	29 c.c.	19 c.c.		39 c.c.
4:30 P.M.	33 c.c.	57 c.c.	10 mg.	35 c.c.
5:30 P.M.	39 c.c.	27 c.c.		31 c.c.
6:30 P.M.	29 c.c.	42 c.c.	10 mg.	29 c.c.
7:30 P.M.	36 c.c.	21 c.c.		27 c.c.
Total	351 c.c.	367 c.c.		372 c.c.
Per cent increase = 4.33 per cent				

HOUR	IX CONTROL (DAY)	IX TREATMENT (DAY)	IX CONTROL (DAY)
	Basal =	Basal =	Basal =
8-9 A.M.	20 c.c.	17 c.c.	29 c.c.
10 A.M.	32 c.c.	10 c.c.	27 c.c.
11 A.M.	33 c.c.	15 c.c.	22 c.c.
12 M.	34 c.c.	10 c.c.	22 c.c.
1 P.M.	31 c.c.	9 c.c.	26 c.c.
2 P.M.	26 c.c.	10 c.c.	23 c.c.
3 P.M.	24 c.c.	19 c.c.	20 c.c.
4 P.M.	24 c.c.	19 c.c.	20 c.c.
5 P.M.	27 c.c.	11 c.c.	20 c.c.
6 P.M.	29 c.c.	13 c.c.	29 c.c.
7 P.M.	--	--	--
Total	260 c.c.	116 c.c.	238 c.c.
Per cent reduction = 55.34 per cent			
Average total reduction for the 9 experiments = 31.2 per cent			

continuous intravenous injection of secretin. Various amounts of ephedrine were injected intravenously and subcutaneously. It was noted, as can be seen on the kymograph tracings (Fig. 1), that 4 mg. of ephedrine, either subcutaneously or intravenously, would produce an inhibition of secretion of pancreatic juice. Occasionally as little as 2 mg. of ephedrine would produce an inhibition (Fig. 2).

Early in the chronic fistula work, 10 mg. of ephedrine were injected subcutaneously every 4 hours; however, it became evident that injections should be made every 2 hours in order to obtain a more satisfactory decrease in secretion. On several occasions the injections were varied slightly, but these variations are shown in Table I.

The summarized results presented in Table I show that a total of 9 experiments carried out on 6 dogs is reported. In every case except 1 the ephedrine produced a definite decrease of the secretion of pancreatic juice. This decrease varied from a low 15.75 per cent to a high 55.34 per cent reduction over the previous control day. The average per cent total reduction for the 9 experiments was 31.2 per cent for the 24-hour period. The one exception found in Part VIII of Table I is easily understood when the basal secretion of 29 c.c. for the control day is compared with the basal secretion of 62 c.c. for the test day. Further, it should be noted that some of the hourly secretions are very high, comparatively speaking, even though the response to ephedrine was very good.

It was also noted that the ephedrine exerted its maximum inhibitory effect during the first hour following the injection, the secretion usually returning to the basal or near basal level by the end of the second hour. To obtain a maximum effect, the ephedrine should probably be injected every hour. No toxic side actions were noted when the ephedrine was injected every two hours, but in contradistinction the animals seemed to be more alert and lively.

The animals survived the experimental procedure from sixteen to thirty days. The appetite usually remained good up until the last day; there was little or no vomiting; and, even though the pancreatic juice was returned via the intestinal fistula and normal saline solution administered subcutaneously, the animals all lost weight. At autopsy the findings were negative except for small hemorrhagic and hyperemic areas found in the pancreas; however, this was not a constant finding. The liver, peritoneum, stomach, intestine, heart, and lungs were all grossly normal. The pH of the blood was within normal limits or very slightly lowered. Death was certainly not explainable, especially from the findings at autopsy.

DISCUSSION

The results clearly indicate that 10 mg. of ephedrine given subcutaneously decrease the volume output of pancreatic juice. This observation agrees with the action of epinephrine on pancreatic secretion.[21, 22] Like epinephrine, ephedrine probably produces its inhibitory

effect on the secretion of pancreatic juice through its vasoconstrictor action on the blood vessels of the pancreas, which results in a decrease in the minute volume flow of blood to the pancreas and a decrease in secretion of pancreatic juice.

From the kymographic tracings, it was determined that the ephedrine begins to exert its vasoconstrictor action about 10 to 15 minutes after injection and continues to exert its effect for the next 20 or 30 minutes; which explains the decrease in secretion noted during the first hour over that of the second hour as observed in the chronic work carried out in the nine experiments as reported in Table I.

Ephedrine in the doses used to decrease the secretion of the pancreas produced no toxic manifestations in the animals. The minimum lethal dose for the dog is 70 mg. per kilogram according to Chen.[25] The average weight of the dogs used in this experiment was 20 kilo.

It is believed that ephedrine is a drug which should be tried to reduce the pancreatic secretion in pancreatic fistula cases. The amount of ephedrine that would be required to reduce the secretion of the pancreas in man cannot be stated; however, a single dose of 400 mg. or the administration of 150 mg. every 3 or 4 hours during the day and night has produced no serious results.[26]

The apparently unexplainable death of the animals with complete pancreatic fistula has been the object of considerable study. Elman and McCaughan,[27] Elman and Hartman,[28] and Bottin[29] have studied the cause of death in these animals. Elman believes that dehydration with resultant changes in circulation are the important factors. Bottin[29] reports that he was unable by returning the pancreatic juice, paraoral fluids, and excellent care of the animals to keep them alive longer than 5 to 7 weeks.

The evidence would appear to indicate that complete pancreatic fistula in dogs without treatment is fatal within from 5 to 8 days.[33] With the oral administration of pancreatic juice, there is a definite reduction in the amount of secretion by the pancreas and animals may be maintained for several months. It is reasonable to say that they may be maintained indefinitely; however, in the experience of Mann and his co-workers, some complication usually arises with the fistula within a few months and the animal survives, but is no longer qualified to pass as an animal having a complete pancreatic fistula. Usually the fistula becomes infected or a new channel establishes itself so that pancreatic juice reaches the intestine and in either case the flow of pancreatic juice through the external fistula diminishes and may soon cease.[32]

The intravenous use of saline or modified Ringer's solution will enable the animals with pancreatic fistula to survive for about 2 months or perhaps longer. The diets of such animals consisted chiefly of meat with the addition of milk or syrup.[32] By the administration of large amounts of alkalies, animals have been kept alive for as long as 16 months.[34]

When pancreatic juice is returned to the intestine, the condition is similar to that of an incomplete pancreatic fistula and such an animal should survive indefinitely; however, some complications usually arise, especially infection of the pancreatic juice, which occurs easily, and the animals survive only a period of a month or so.

The problem concerning complete pancreatic fistula, therefore, raises many questions which cannot as yet be answered with certainty.

Prevention of Skin Erosion.—Patients with pancreatic fistula, especially those draining activated pancreatic juice, present a serious problem in the management of the skin and tissues of the abdominal wall. This is because the skin and tissues with which the juice comes into contact are markedly irritated, digested, and occasionally large deep ulcers are formed.

Silica gel has proved to be very valuable in controlling the skin excoriation and tissue digestion in two clinical cases.[31] It has been used on the theory that it absorbs enzymes. It is a neutral powder, very soothing, nonirritating, and easily applied. It has been used in these experiments as a dressing powder.

Two of the animals, not reported in this series, leaked pancreatic juice around the tube leading from the pancreatoduodenofistula. Marked irritation of the abdominal wall occurred; however, after several applications of silica gel as a dressing powder, held in place by an abdominal binder, the excoriation and irritation cleared up, and no further irritative changes took place, due to the continued application of the silica gel. On withdrawal of the powder, the irritation returned, to disappear on the resumption of the use of the powder. In all of the animals reported in this series, prophylactic measures were taken, in that silica gel dressings were applied once or twice daily in order to prevent excoriation and irritation of the skin about the jejunal fistula. None of the animals developed any irritative changes even though several of the animals had copious drainage from the intestinal fistula. In duodenal fistula cases it is suggested that the fistulous tract be packed with the gel.

CONCLUSIONS

1. Ephedrine decreases the output of pancreatic juice. In this regard it acts like epinephrine, but it is more convenient to use than epinephrine.

2. Silica gel has been found, when applied freely and frequently, to prevent skin erosion due to activated pancreatic juice.

3. It is believed that the use of ephedrine in the management of patients with pancreatic or duodenal fistulas is indicated; at least, it should be given a trial. Silica gel likewise should be given a trial.

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RECURRENT DISLOCATION OF THE SHOULDER JOINT; AN EVALUATION OF THE NICOLA OPERATION

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THE VARIETY of procedures advocated for the surgical correction of recurrent dislocation of the shoulder joint have been reviewed by Speed,[1] Henderson,[2] and Willard.[3] In the main, uniformly better results have been obtained in this country by operations of the suspension type than by procedures directed to the bony and muscular structures and upon the capsule itself. It has been logically contended that, if the head of the humerus is suspended to the bony arches above, it will be prevented from slipping forward and downward, and recurring dislocations thereby obviated; and this has been substantiated by the clinical experience of surgeons employing suspensions of tendon or fascia, either slung beneath the neck (Carrell,[4] Fowler[5]) or through the neck of the humerus (Joseph,[6] Henderson,[2, 7] Nicola[8-12]).

Nicola, obtaining the idea for his operation from observations on an articulated skeleton in which the humeral head was held in place by a metal pin transfixing it and attached to the upper angle of the glenoid fossa, has recommended that the head of the humerus be suspended within the glenoid cavity by passing the tendon of the long head of the biceps brachii through the head and neck, creating an intra-articular ligament not unlike the ligamentum teres of the hip joint.

Briefly, through a 3-inch incision beginning above, just outside the coracoid process, and dividing the most medial fibers of the deltoid muscle by blunt dissection, the tendon of the long bicipital head is exposed in its groove after severance of the transverse humeral ligament and is divided ½ inch above the pectoralis major, after silk sutures have been placed through its proximal and distal parts. The proximal portion is drawn through a channel in the head and neck of the humerus, made with a ¼-inch drill and is reunited to the distal portion of the tendon with the elbow flexed at 45 degrees and then to the surrounding tissues with the arm abducted 90 degrees. The transverse humeral ligament is repaired, the muscle layer and skin closed, and the arm is immobilized in a Velpeau bandage for 2 to 3 weeks (in epileptics for 6 weeks).

To insure a successful result:

1. The osseous channel must be directed in the normal course of the long bicipital tendon, beginning in the bicipital groove about ½ inch

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below the lesser tuberosity, and appearing on the surface of the head ½ to ¾ inch from its articular margin. If less than ½ inch, redislocation may occur because the tendon will not check the arm in extreme abduction.

2. The tendon must become firmly fixed in the tunnel or it will fail to check redislocation and may fray or even tear with constant friction. Therefore, the tendon, divested of synovial membrane, must fit snugly in its channel and must be fixed in the bicipital groove by suturing it to the adjacent periosteum and transverse humeral ligament.

3. The arm is abducted 90 degrees after the severed ends of the tendon are sutured and prior to fixation to surrounding tissues to insure enough tendon between the glenoid cavity and humeral head so that normal abduction might not be restricted.

Nicola[12] in 1935 reported his series of 37 cases (now increased to 57)[13] with 1 recurrence following a violent injury. Three recurrences following the use of his method had been reported to him, and reoperation in 2 revealed that the channel had been too large for the tendon in one and in the other that the tunnel had not been constructed far enough on the articular surface to check the head with the humerus abducted 180 degrees. Willard[3] in 1936 reported 10 cases operated upon by the Nicola method, with 1 recurrence 17 months postoperatively following a severe injury, but without further recurrence in the 16-month period thereafter. Other authors[14-16] have reported small series of successful cases.

While the several suspension methods have proved uniformly satisfactory, especially in the hands of their originators, the following survey is made to evaluate the Nicola operation as performed in several clinics by a number of competent surgeons varying in their experience and skill in bone and joint surgery.

ANALYSIS OF CASES

Twenty-five cases of recurrent dislocation of the shoulder joint have been treated by the Nicola procedure. Five are excluded from this study, 1 because of inadequate follow-up data and 4 because they have been operated upon too recently (1 to 5 months) for evaluation, even though none has experienced recurrence.

The authors have combined the Nicola operation with arthrodesis of a paralytic shoulder joint in 1 case, as recommended by Wagoner,[17] so as to secure more adequate internal fixation during fusion.

The remaining 20 cases are analysed in Table I. All were males except 3. The youngest patient was 12 years old; 2 were 17 years; 1 was 19 years; and the remaining 16 ranged from 20 to 28 years in age at the time of operation. The right side was affected in 9, the left in 10, and involvement was bilateral in 1.

TABLE I

ANALYSIS OF 20 CASES OF RECURRENT DISLOCATION OF THE SHOULDER JOINT

NAME	AGE	SEX	SIDE	NO. OF DISLOCATIONS	DURATION UP TO NICOLA OPERATION	DURATION FOLLOWING OPERATION	RESULT
J. P.	22	M	L	13	3 yr.	11 mo.	No recurrence; normal shoulder joint motion
C. L.	19	M	R	10	3 yr.	1 yr.	No recurrence; normal shoulder joint motion
C. M.	22	F	L	30 to 40	6 yr.	1 yr. 8 mo.	No recurrence; normal shoulder joint motion
D. B.	17	M	R	"Many"	6 yr.	8 yr.	No recurrence; normal shoulder joint motion
J. W.	20	M	R	15	2 yr.	5 yr. 5 mo.	No recurrence; normal shoulder joint motion
A. S.	17	M	R	6	2 yr.	4 yr.	No recurrence; normal shoulder joint motion
C. U.	20	M	R	"Many"	2 yr.	1 yr. 6 mo.	No recurrence; normal shoulder joint motion
F. J.	21	F	L	"Many"	3 yr.	3 yr.	No recurrence; normal shoulder joint motion
S. H.	25	M	L	4	3 yr.	3 yr.	No recurrence; normal shoulder joint motion
C. N.	26	M	L	"Many"	6 mo.	2 yr. 5 mo.	No recurrence; normal shoulder joint motion
O. C.	25	M	L	"Many"	4 mo.	2 yr. 6 mo.	No recurrence; normal shoulder joint motion
R. M.	28	M	L	"Many"	11 yr.	10 mo.	No recurrence; normal shoulder joint motion
H. F.	21	M	L	25	2½ yr.	1 yr. 7 mo.	No recurrence; normal shoulder joint motion
S. M.	22	M	R	3	4 mo.	6 mo.	No recurrence; normal shoulder joint motion
M. R.	22	M	L	3	6 mo.	2 yr. 3 mo.	No recurrence; normal shoulder joint motion
E. T.	20	M	R	"Many"	1 yr.	7 yr. 5 mo.	No recurrence; normal shoulder joint motion
M. S.	22	M	L	4	2 yr.	7 yr. 6 mo.	No recurrence; normal shoulder joint motion

TABLE I—CONT'D

NAME	AGE	SEX	SIDE	NO. OF DIS-LOCATIONS	DURATION UP TO NICOLA OPERATION	DURATION FOLLOWING OPERATION	RESULT
J. H.	24	M	R	3	2 yr.	1½ yr.	1½ yr. free period; recurrence following severe injury; Henderson type secondary repair
H. M.	12	F	R	3	1 yr.	5 yr. 8 mo.	6 mo. free period; recurrence without injury; preternatural motion of humeral head; marked hiatus in anterior muscle wall; x-ray shows deformed head; humeri of equal length
J. C.	21	M	Bil.	R-Over 200 L-20 to 30	1½ yr. 6 mo.	R- 7 mo.	8 mo. free period; recurrence of "slipping" sensation, without injury; normal shoulder joint motion

In each case the initial dislocation had been due to a severe injury, but a complicating fracture (head of the humerus) occurred in only 1. There was 1 epileptic, in whom the initial discolation occurred during a convulsion. It is noteworthy that in the last 5 cases specifically interrogated, 3 had been immobilized following the initial dislocation for only one week or less.

The number of redislocations varied from 3 to "200 or more," over a period ranging from 4 months to 11 years. Each gave a history of increasing frequency of redislocation and ease of reduction. In one patient so slight an effort as sneezing initiated a luxation. During the free periods they experienced no discomfort and, except for occasional atrophy of the shoulder muscles, the involved joint was apparently "normal." Roentgenograms taken prior to operation were negative for congenital or traumatic defects.

In only 1 case had previous operation been attempted, an unsuccessful capsulorrahy.

The Nicola technique was carefully adhered to. Fixation was usually obtained with a Velpeau bandage, and in 2 cases by means of a plaster shoulder spica, for 2 to 3 weeks, followed by physical therapy and guided exercises. The epileptic was immobilized for 6 weeks.

The period since operation varies from 6 months to 8 years; from 6 to 11 months in 4, 1 to 2 years in 5, 2 to 5 years in 6, and over 5 years in 5.

There have been no recurrences in 17 of the 20 cases over periods varying from 6 months to 8 years.

While 3 of these "successful" cases have been observed for a period of less than 1 year, the fact that they had dislocated, prior to operation, "13 times in 3 years," "3 times in 4 mos.," and "many times each year for 11 years," is obviously significant.

J. H., after a 1½-year period free of luxation, suffered recurrences following a severe injury to the affected shoulder joint. An exploration revealed that the portion of the tendon spanning the joint had been ruptured, and a suspension procedure of the Henderson type was performed. There has been no recurrence since.

In case H. M. dislocations recurred without injury after a free period of 6 months. This patient feels that she has been benefited in that the shoulder feels stronger and is not habitually dislocated as it was prior to operation. She refuses to undergo a second repair.

In case J. C. the occurrence of redislocation, without injury, after a free period of 7 months, is questionable until proved clinically and by stereoscopic roentgenograms. The patient describes a "slipping sensation" which may be an entirely subjective symptom. The development of his adductors and internal rotators is being encouraged by exercises.[18] The surgeon in this case feels that he may have erred in failing to construct the bony channel more than ½ inch from the articular margin of the humeral head.

DISCUSSION

It is probable that the usual underlying pathologic process in recurrent dislocation of the shoulder joint is an unduly relaxed anterior and inferior joint capsule. In the reported cases the initiating factor has almost invariably been traumatic. The relative frequency of this condition suggests the need for more adequate and prolonged fixation in the acute type of shoulder dislocation and the avoidance of too early and overzealous abduction and external rotation.

Although we have had no personal experience with the plan suggested by Davis[18] as a conservative treatment for this chronic disability, we feel that the institution of such a regime also during the treatment of the acute (initial) shoulder dislocation might prove a valuable prophylactic measure; i.e., the development of the adductor and internal rotator muscles of the shoulder, so as to fortify the anterior aspect of the joint and so that its important check strap, the long bicipital tendon, might exert its normal effect backward and medialward against the humeral head.

The reported recurrences have followed either faulty technique in the creation of the osseous channel and in the fixation of the tendon, or a severe injury to the reconstructed shoulder joint. In our case, J. H., such a severe trauma resulted in rupture of that portion of the long biceps tendon spanning the joint cavity between the glenoid cavity and the humeral head.

Treatment of cases in which the long bicipital tendon cannot be utilized requires some other suspension procedure, as of the Joseph-Henderson or Carrell-Fowler type. While the short head of the biceps, detached from the coracoid process and passed through the greater tuberosity and acromion process, has been used by Nicola,[13] recent studies by one of us (M. T. H.) at the Daniel Baugh Institute of Anatomy, Philadelphia, indicate that the thick tendon of the short head will not reach this entire course and must be supplemented by additional tendon or fascia.

The failure in H. M., aged 12 years, may be ascribed to several factors. A marked hiatus, present between the deltoid and pectoralis major muscles, and due to unnecessary stripping of the former from the clavicle and its inadequate repair, has further impaired the already defective anterior portion of the shoulder joint. The preternatural mobility of the humeral head, in the absence of injury, indicates excess length of the tendon between the humeral head and the glenoid fossa, due either to stretching or to improper construction of the osseous channel. However, the deformity of the head of the humerus in this case, secondary to growth disturbance following penetration of the epiphyseal disk, may have further disturbed the mechanics of the new intra-articular ligament and assisted in recurrence. The Nicola operation should be deferred until active growth at this disk has ceased (seventeenth year or later).

CONCLUSIONS

The Nicola operation, properly executed, corrects the disability of a recurrently dislocating shoulder joint. Failures are to be anticipated where the proper technique is not adhered to or when overly severe trauma is inflicted upon the translocated tendon of the long head of the biceps brachii muscle.

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SURGICAL TREATMENT OF STRICTURE OF THE RECTUM

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THE satisfactory treatment of inflammatory strictures of the rectum is difficult and frequently necessitates permanent interruption of the normal intestinal continuity. In the following case a segmental resection of the rectum which preserved intestinal continuity was carried out successfully. We believe this operative procedure is an alternative to be considered in certain similar cases.

REPORT OF CASE

The patient, a married woman 31 years of age, came to the clinic Aug. 3, 1936, having been referred because of a stricture of the rectum. She said that she had been well until 1926, when a rather acute "enteritis" had developed. At this time she was constipated and bowel movements were associated with the passage of considerable pus and some blood. She was treated for this by multiple enemas and by conservative medical procedures. The acute phase readily subsided, but a rather persistent constipation and some pain on defecation remained. A small tumor, the description of which we were unable to obtain, was then removed from the rectum in 1929. Following this, the patient continued to have constipation. There was an increasingly severe pain with each bowel movement, but at no time was there gross bleeding from the rectum. In August, 1934, the patient was told that she had multiple ulcers and considerable inflammation in the rectum. She consulted another physician a few months later and was given several radium and roentgen treatments to the rectum. Following this, the pain subsided, but obstipation became more marked. The caliber of the stools decreased to the size of a thin ribbon and in an attempt to get relief the patient developed the habit of using cathartics. At times a small amount of mucopurulent discharge came from the rectum, but no gross bleeding was noted. The patient's trouble became most marked in the twelve-month period preceding admission. She had lost 22 pounds (10 kg.) during this period.

Nothing of importance was noted on general examination at the clinic other than the rectal stricture. Rectal examination revealed a tense sphincter and a rather soft, fibrotic-feeling stricture which could easily be palpated in the middle portion of the rectum (Fig. 1*a*). Proctoscopic examination showed this stricture to begin 6 cm. above the anus, to be 2.5 to 3 cm. in length, and to have a lumen of less than 0.7 mm. in diameter. A Wassermann test for syphilis was negative. The Frei test, using the Frei antigen, was also negative. The value for hemoglobin was 13.6 gm. per 100 c.c., and erythrocytes numbered 4,240,000 and leukocytes 9,000 and 10,300 per cubic millimeter of blood. A diagnosis of inflammatory stricture of the rectum was made and, inasmuch as the bowel above and below the stricture seemed normal, it seemed advisable to attempt local excision of the stricture.

On May 11, 1936, loop colostomy was performed through a left McBurney incision, the abdomen being explored at the same time. No evidence of malignancy was felt, but in the cul-de-sac one could feel a little increased resistance. The patient made an uneventful recovery and was dismissed from the hospital fifteen days after admission.

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On June 11, 1936, under spinal anesthesia, segmental resection of the strictured portion of the rectum was performed. The patient was placed in the dorsal decubitus position and a Kraske incision was made. The coccyx was so frozen that it was necessary to remove it with a chisel. None of the sacrum was removed. There was considerable induration of the fat immediately around the involved portion of the rectum. The rectum was loosened and the peritoneal cavity was opened. The rectosigmoid was pulled down until it was entirely extraperitoneal and the peritoneum was closed around it (Fig. 1*b*). A segment about 10 cm. long was removed from the middle portion of the rectum. An end-to-end anastomosis was made over a large rectal tube, using chromic catgut and silk sutures (Fig. 1*c*). The wound was separated from the anastomosed bowel by a piece of rubber sheeting. Four Penrose drains were inserted and the wound was closed loosely with interrupted sutures. The rectal tube was left in situ. The pathologist reported the resected portion of the rectum to contain an inflammatory stricture. Microscopically, a large number of mast cells were found to be present.

The patient had a relatively uneventful convalescence and was dismissed from the hospital on the forty-second day. The temperature was between 103 to 104° F. during the first few postoperative days and then gradually subsided in two weeks

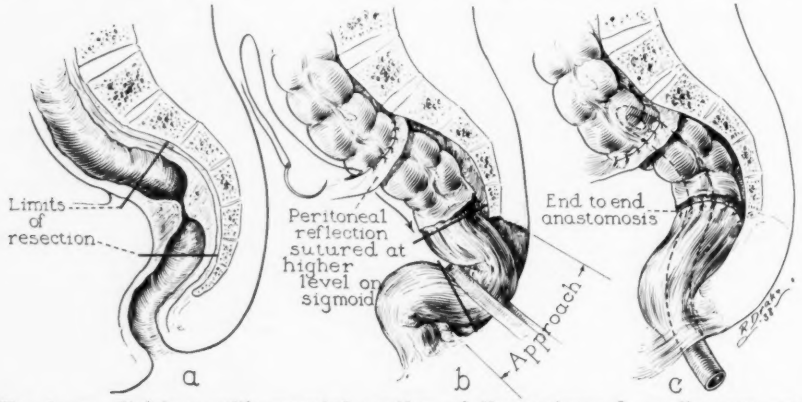

Fig. 1.—*a*, Stricture with resected portion of the rectum; *b*, peritoneum opened, rectosigmoid pulled down, and peritoneum closed; and *c*, end-to-end anastomosis of rectum over a rectal tube.

to normal. The drains were all removed and the rectal tube was changed on the tenth postoperative day. Daily Sitz baths were begun and the patient was allowed to be out of bed.

The patient was temporarily dismissed from the clinic on the forty-seventh postoperative day. At this time her general condition was good. There was a small amount of seropurulent drainage from the posterior wound, but it was clean and granulating rapidly. Proctoscopic examination showed the lumen of the rectum to be about 4 cm. in diameter.

The patient did not return to the clinic for closure of the colonic stoma until one year later, although she was repeatedly advised to do so. At this time the bowel was negative as far as the proctoscope could reach. The lumen of the rectum at the site of the anastomosis was 3 cm. or more in diameter. A small sinus posterior to the anus drained a small amount of purulent material. This was curetted and repeatedly packed, allowing it to granulate gradually. Clamps were applied to the spur of the colonic stoma and, on Aug. 17, 1937, extraperitoneal closure of the stoma was performed. The patient was dismissed from the clinic Sept. 1, 1937. At this time the abdominal wound had healed, the rectum was ample, and the area of the anastomosis easily admitted two fingers. The patient was having normal bowel movements and had no symptoms referable to the rectum. She has remained well since.

COMMENT

An inflammatory, simple, or fibrous stricture, such as was seen in this case, is the result of contraction of scar tissue caused by some injury or severe inflammation in the rectal wall or surrounding tissues. This may be the result of accidental or operative trauma, of a localized ulcer or abscess, or of some general inflammation of the rectum and surrounding tissues. All forms of septic inflammation may be responsible for the condition, but certain rather specific types, such as gonorrheal or syphilitic inflammation or lymphopathia venerea, frequently are found.

The type of tumor originally removed in this case is not known, but it seems reasonable that it was this operative procedure that removed an area of the mucous membrane. This then probably left an ulceration which became septic with some surrounding inflammation, and perhaps even secondary ulcer formation. The healing of this ulcerative process would in itself have been sufficient to cause the formation of a fibrous stricture. However, in addition, radium and roentgen therapy were employed, and this was also a probable factor in the formation of the stricture.

The original cause of the stricture is not as a rule of great concern to the surgeon because he is called to treat the late result of a long-standing inflammation. It is only after a long period of chronic inflammation, perhaps of several years' duration, that the stricture becomes severe enough to require operative interference.

It probably can be said that the majority of inflammatory rectal strictures do not produce sufficient symptoms to require treatment. It is only when the stricture causes definite obstruction to the passage of feces that symptoms develop. In these cases the choice of treatment depends on the location, type, and extent of the stricture. Obviously it is desirable to know whether the stricture is at the anus, in the lower part of the rectum below the peritoneal reflection, or higher. One should know whether or not he is dealing with an annular or tubular stricture and what the condition of the bowel is above and below this region. It is the more or less universal opinion that the best treatment in the very mild cases is gradual dilatation; in the more severe cases, proctotomy; and in the extremely severe cases, establishment of a permanent colonic stoma. Every case, however, should be analyzed as an individual problem in which some form of excision or resection of the rectal stricture can be done.

The operation which was performed in our case offers another method of resection in certain cases of inflammatory stricture of the rectum. It is desirable that the patient be young and a good surgical risk, that the stricture be located in the midrectum well below the peritoneal reflection, and that the bowel above and below the stricture be in good condition. It is advisable to perform preliminary colostomy in an attempt to put the rectum at rest and to enable one to keep the parts sufficiently free from gross contamination to render the operation safe.

BILE SALTS IN THE TREATMENT OF PEPTIC ULCER

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NUMEROUS observations from the laboratory and from the clinic have indicated that the liver and its secretions may be related in some way to the etiology of peptic ulcer.

It is well known that spontaneous duodenal or gastric ulcer is often associated with hepatitis and liver insufficiency in the dog. Ulcers may develop in dogs with common duct obstruction,[2, 4, 7, 17-19] with external[2-4, 15, 16, 22, 34] or internal biliary fistula,[10, 11, 14, 21, 24, 27-29, 32, 37, 49] following plastic operations upon the bile ducts,[17, 20] after establishment of an Eck fistula[6, 19] or other interference with the hepatic blood supply,[18] after partial hepatectomy,[6] or following the administration of certain drugs which damage the liver.[9, 20, 39, 44, 45, 47]

Observations of peptic ulcer associated with liver disease or disturbed hepatic physiology in man have also been reported.[8, 25, 26, 35, 38, 41, 42, 48] Furthermore, Schnitker and Hass[38] have reviewed microscopic sections of the liver in 100 post-mortem cases of peptic ulcer and in 100 controls and have found that there were more ulcer cases than control cases which exhibited liver damage, and that the livers from patients with ulcer showed more marked evidence of pathology than did those of the controls.

With these facts in mind, a clinical study has been carried out during the past year in order to determine the effects of the administration of bile salts upon the course of the disease in patients with gastric or duodenal ulcer.

METHOD

This report deals with the treatment of a group of 32 cases of duodenal (26 cases), gastric (5 cases), or gastrojejunal (1 case) ulcer by the oral administration of iron bile salts* (Bilron-Eli Lilly and Company). In every case the diagnosis was established by roentgenoscopic and roentgenographic examination, and in 4 of the 5 cases of gastric ulcer gastroscopic examination was also carried out. In 20 of the 26 cases of duodenal ulcer activity of the disease was indicated by the demonstration of a niche by the roentgenologist, and the remaining 6 cases as well were active as judged by clinical symptoms. The condition of the gall

*The Bilron used in this study has been supplied through the courtesy of Eli Lilly and Company.

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bladder was also investigated by cholecystographic examination in 17 cases, and in all of these except 1 (Case 26) the gall bladder was found to be normal.

All patients were ambulant during treatment, and no attempts were made toward selection except that patients with perforation or massive hemorrhage, of course, were not included. Most of the patients in this series have been under our observation for periods of from six months to one year or more.

Patients referred to the University Hospital Clinic have all been seen previously by other physicians and many of our group of cases, as can be judged from Table I, had received some form of treatment before the bile salt therapy was instituted. At the time of instituting the bile salt therapy, however, all patients were suffering from symptoms of their disease. Seventy-five per cent of the patients had suffered from ulcer symptoms for three years or more. There were 22 males and 10 females in the group.

Since most patients had previously been following a restricted diet, they were instructed to return to a general diet when the bile salt therapy was started. A few of those with the most severe symptoms were retained on a somewhat restricted regimen, but even the latter were allowed more latitude than on the diets which they had previously been following. Case 30 was an exception to this general rule. He was placed on a strict schedule of hourly bland feedings in addition to the bile salt therapy, but he was not improved and operation was subsequently required.

The iron bile salts were given in 10-gr. doses four times a day—with each meal and at bedtime. In a few cases this amount, 40 gr. per day, produced an undesirable catharsis and in those cases the dose was cut to the limit of tolerance.

RESULTS

The results of the administration of bile salts to the 32 patients with proved duodenal, gastric, or gastrojejunal ulcers are listed briefly in Table I. Twenty-six patients, or 81.25 per cent, were definitely improved (Cases 1-26), while 6 patients, or 18.75 per cent, were not improved. Many of the patients who had suffered from ulcer symptoms for years were highly gratified because of the improvement experienced.

We learned early that some patients develop *increased pain* for a few days after starting to take bile salts, so all were warned of this possibility. Ten of our 32 patients experienced increased distress for periods of three or four days up to two weeks. We have assumed that this may be due to a local irritative action of the bile salts.

A week or ten days after starting the therapy, sometimes more and sometimes less, improvement usually became manifest and gradually progressed until many patients felt entirely well. They soon found that

TABLE I

CASE	AGE	SEX	BRIEF CASE HISTORY	PRESENT DIAGNOSIS	RESULT OF BILE SALT THERAPY	CHOLECYSTOGRAM
1	38	M	18-year history of ulcer symptoms; present recurrence of symptoms of 2 or 3 months' duration	Duodenal ulcer with deformity of duodenal bulb	Improved General diet Could do hard work	Negative
2	21	M	Had had diagnosis of gastric ulcer in 1934; recurrent symptoms for 2 years, increasing in severity; had been confined to bed for 3 weeks, taking restricted diet and antacids; 20-pound weight loss recently	Duodenal ulcer with crater	Improved Working General diet	-
3	22	M	Ulcer symptoms since 1935; no previous therapy	Duodenal ulcer with crater	Improved General diet	-
4	64	M	Epigastric distress for 1 year; severe during past 3 weeks	Large duodenal ulcer with niche	Improved General diet	Negative
5	36	F	Symptoms of ulcer for several years; had been on Sippy regime since 1934; worse recently	Duodenal ulcer with crater	Improved General diet	Negative
6	45	F	Ulcer history since 1928; had been on regime of diet and alkalies, but still suffered from symptoms	Old duodenal ulcer with marked scarring	Improved	Negative
7	27	M	Ulcer symptoms for 6 years, becoming worse; recently had been unable to work, and had been on restricted diet, chiefly milk	Duodenal ulcer with crater	Improved Working part time General diet	-
8	21	M	Patient had been in hospital 1 month previously for bleeding duodenal ulcer, where he had been treated with bed rest, diet, and alkalies; after 3 weeks of treatment he was discharged with a dietary regime; 1 month later bile salt therapy was started	Duodenal ulcer with niche	Improved Doing heavy farm work General diet	-

TABLE I—CONT'D

CASE	AGE	SEX	BRIEF CASE HISTORY	PRESENT DIAGNOSIS	RESULT OF BILE SALT THERAPY	CHOLE-CYSTO-GRAM
9	38	M	Epigastric pain for 2 years; so severe that he had been unable to work during the past 1½ years	Duodenal ulcer with crater	Improved Doing heavy work General diet	Negative
10	36	M	Ulcer symptoms for 8 years; operation for perforated ulcer in 1936; recurrence of pain for past 8 weeks	Duodenal ulcer with crater	There was a rapid improvement, but five months later, there was a slight relapse, for which the patient took alkalies for two weeks; there was a rapid recovery from the relapse	-
11	34	M	Ulcer symptoms for several years; gastroenterostomy in 1932; recurrence of ulcer symptoms	Gastric ulcer on lesser curvature; also old deformity of duodenal bulb	Improved Increased diet	-
12	33	F	Epigastric distress for 15 years; aggravated the last 2 years; had been in hospital for 5 weeks with a regime of bed rest, diet, and alkalies, with some improvement; re-examination after 3 months revealed a large crater still present; bile salt therapy was then started and the diet was increased; re-examination 4 months later revealed the crater to have disappeared	Duodenal ulcer with niche	Improved General diet	Negative
13	30	M	8-year history of ulcer symptoms, recently becoming worse; patient had eaten nothing but milk for 3 weeks	Duodenal ulcer with niche	Improved General diet Moderate work After four months the patient had a mild relapse which lasted 6 days, but from which he rapidly recovered	-

TABLE I—CONT'D

CASE	AGE	SEX	BRIEF CASE HISTORY	PRESENT DIAGNOSIS	RESULT OF BILE SALT THERAPY	CHOLECYSTOGRAM
14	22	M	Epigastric pain and right lower quadrant soreness for several weeks; no previous therapy	Duodenal ulcer with large crater	Improved; the patient was clinically well, but x-ray still showed a small crater after four months General diet	-
15	54	M	Ulcer symptoms for 30 years; severely aggravated the past 3 months; patient had been in bed for several weeks and had been on a very restricted diet	Duodenal ulcer with crater	Improved General diet	Negative
16	46	M	History of ulcer symptoms for 15 years; becoming much worse the past few months; hemorrhages in 1922 and 1924; had previously been on Sippy regime	Duodenal ulcer with niche	Improved General diet Light work	Negative
17	55	F	Epigastric pain for 15 years, but much worse the last 3 years; had been on restricted diet	Gastric ulcer on lesser curvature	Improved General diet	Negative
18	44	F	Epigastric distress for 1 year; no previous treatment	Duodenal ulcer with niche	Improved General diet	Negative
19	60	M	Ulcer symptoms for 15 years; had been treated with Sippy regime; severe recurrence of pain for several weeks with vomiting the past week	Gastric ulcer on lesser curvature	Improved Ulcer healed General diet	-
20	33	M	Periodic epigastric distress for 7 years; worse the past 6 months; unable to work; had been on Sippy regime	Duodenal ulcer with niche	Improved General diet Moderately heavy work	Negative
21	75	M	Long history of ulcer symptoms, with recent aggravation	Duodenal ulcer with niche	Improved General diet	-
22	40	M	Ulcer symptoms for 8 years; had been previously treated with a mucin preparation	Duodenal ulcer with niche	Improved General diet	Negative

TABLE I—CONT'D

CASE	AGE	SEX	BRIEF CASE HISTORY	PRESENT DIAGNOSIS	RESULT OF BILE SALT THERAPY	CHOLECYSTOGRAM
23	35	M	Epigastric distress for 10 years, becoming worse the past few months; had been on a restricted diet	Duodenal ulcer with deformed bulb, but no crater	Improved General diet	-
24	41	F	Patient had had ulcer symptoms for many years; she had had a gastroenterostomy in 1929, but the ulcer symptoms soon recurred; had been hospitalized twice during the past few months because of hemorrhage from a duodenal ulcer; was on a regime of diet and alkalies, but still suffered considerable discomfort; institution of bile salt therapy followed by rapid improvement	Duodenal ulcer with crater; gastroenterostomy	Improved General diet except for a few slight restrictions	-
25	51	F	Epigastric distress, which had been becoming worse during the past 2 years; had been treated with synodal	Duodenal ulcer with deformed duodenal bulb	Improved General diet	Negative
26	56	F	Ulcer symptoms for 10 years; she was first placed on Sippy regime plus bile salts; six months later the alkalies were discontinued, the dietary restrictions were removed, and patient was given only bile salts	Duodenal ulcer with niche	Improved	Pathologic gall bladder
27	50	M	Patient had previously had perforated ulcer on two occasions, requiring operation; at last operation, 1 year ago, gastroenterostomy had been done in addition to closure of perforation; symptoms soon recurred, and at the present time the patient was having severe distress	Jejunal (stoma) ulcer	No improvement from Bilron therapy for 4 weeks; patient then placed on Sippy regime with some improvement	-

TABLE I—CONT'D

CASE	AGE	SEX	BRIEF CASE HISTORY	PRESENT DIAGNOSIS	RESULT OF BILE SALT THERAPY	CHOLECYSTOGRAM
28	36	M	Ulcer symptoms for many years; for past few months had suffered marked obstructive symptoms, and had obtained no relief, dietary treatment and bed-rest in a hospital; no relief from bile salts; probably will require operation	Duodenal ulcer with obstruction	No improvement after 6 weeks of Bilron therapy	–
29	53	M	History of periodic ulcer distress for many years; much worse during the past two years	Duodenal ulcer with niche	Temporary improvement for 3 months; there was then a recurrence, and the follow-up is incomplete	Negative
30	50	M	Patient had a huge gastric ulcer which did not respond to bile salt therapy used in conjunction with hourly bland feedings for seven weeks; anterior gastroenterostomy was subsequently performed, and this was followed by marked improvement	Large gastric ulcer	No improvement; operation was required	Negative
31	48	F	Patient had been on a regime of diet and alkalies periodically for 8 years for relief of symptoms of duodenal ulcer	Gastric ulcer on lesser curvature, and chronic duodenal ulcer	Patient felt about the same as when on the Sippy regime	Negative
32	59	F	Epigastric pain relieved by food, of 2 months' duration; bile salt therapy was followed by very slight improvement; after a 4-month trial, the bile salt therapy was discontinued and the Sippy regime was tried with approximately the same result	Chronic duodenal ulcer without niche	Very little improvement	Negative

they could eat many foods which had previously caused them distress, and they were glad to return to a general diet. This statement, however, must be qualified to a certain extent, since many patients still found that the ingestion of very greasy or highly seasoned foods might be followed by some degree of discomfort. It also was possible for patients to resume work of a moderately strenuous nature, and in some cases the patients were capable of returning to very heavy labor.

X-ray evidence of healing came more slowly than the clinical improvement, and in the early follow-up examinations it was not unusual to find a niche still present, in spite of the fact that the patient was feeling well, was taking a general diet, and had returned to work. Later, however, the niche diminished or vanished, giving roentgenologic evidence of healing in conformity with the clinical improvement.

COMMENT

The mechanism by which the bile salts exert a beneficial effect in cases suffering from ulcer is obscure. There is no permanent alteration of gastric acidity,[23] although it is possible that the increased flow of bile which is known to be stimulated by the administration of bile salts may result in a greater regurgitation of duodenal contents over the ulcer-bearing area so that the acid gastric juice may be more effectively buffered. It is well known that operations designed to deviate the bile flow into the stomach for the purpose of neutralizing the acidity of the gastric juice have not only failed to influence ulcers beneficially, but, on the contrary, cholecystogastrostomy or cholecystoduodenostomy has been followed occasionally by the spontaneous development of ulcers,[37] presumably as a result of the hepatic damage from ascending infection. Such an unphysiologic arrangement, however, cannot be compared to our method of treatment, since anastomotic operations upon the biliary tract are invariably followed by the development of an ascending infection.[37] Furthermore, if the factor of regurgitated duodenal juices is really important as a protective mechanism, the increased volume of bile may be beneficial from a mechanical standpoint, by carrying with it pancreatic juice and succus entericus, which have a buffering capacity in addition to that of bile.

Although such an explanation would seem to explain most simply the beneficial action of the bile salts, there are other possible mechanisms which should be considered. It is possible, although it does not seem probable, that the bile salts might exert some local effect stimulating healing. It is also possible that the bile salts supply, or stimulate the liver or some other organ to supply, some unknown deficient factor which might be important in the protection against the development of ulcer. Finally, the bile salts may aid in the relaxation of local spasm which, according to some investigators, may be a factor in the etiology

of ulcer. It is generally agreed that bile salts stimulate activity of the large bowel, but there is some evidence to indicate that they might inhibit the motility of the small intestine.[43]

Blanck[4] and Ochsner, Gage, and Hosoi[30] have found whole bile to exert a protective influence in animals under experimental conditions tending to produce ulcer. Bollman and Mann,[7] on the other hand, failed to prevent ulcer in dogs by the oral administration of gall-bladder bile twice daily following ligation of the common bile duct.

Emery and Schnitker[13] recently have reported the results of a clinical study of the effect of administering a specially prepared desiccated bile to 40 patients suffering from peptic ulcer. They found that many of the patients experienced definite relief and concluded that, while the results were such as to exclude it as a specific treatment for the disease, bile does have a beneficial effect which does not seem readily explicable by a lowering of gastric acidity. It is apparent that these findings are in agreement with those observed in our study, which suggests that perhaps the beneficial results obtained by Emery and Schnitker were due to the bile salts present in their preparation. Whether any one of the individual bile salts or bile acids is more effective than others in its favorable action cannot be stated at this time.

Knowing that acute gastric ulcers may be produced in animals by toxic doses of bile salts,[36, 40, 46] one might question the advisability of administering these substances for therapeutic purposes. Such acute ulcers, however, are quite distinct from the type of ulcer that the clinician treats and similar acute ulcers can be produced by toxic doses of many drugs. Furthermore, the ulcer-producing dose of bile salts is greater than the death-producing dose,[46] and one would certainly not expect any unfavorable effects from the doses which we have used. The fact that acute gastric ulcers or erosions do not follow the administration of bile salts according to our method has been established in several cases by gastroscopic examination.

It should be recognized that the cases receiving bile salts in this clinical study have not been compared with a control group receiving an inert substance according to a similar schedule. For this reason, one must be guarded in the conclusions drawn, but the beneficial results following the administration of bile salts seem so definite that it appears to be justifiable to report our uncontrolled series in the hope that other clinicians may be stimulated to assist in finally evaluating this therapeutic method. At the present time, with the cooperation of Dr. John A. Layne, a controlled study is being carried out in this clinic.

SUMMARY

A clinical study has been carried out in order to determine the effects of the oral administration of bile salts (Bilron) upon the course of the disease in patients with gastric or duodenal ulcer. In an uncontrolled

group of 32 patients, definite improvement was noted in 26 cases, or 81.25 per cent. Although it is clear that bile salts do not constitute a specific treatment for ulcer, they do appear to exert a definitely beneficial effect in many cases, and this form of therapy may prove to be a valuable adjunct in the treatment of patients with gastric or duodenal ulcer.

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A DEVICE FACILITATING THE ADMINISTRATION OF CAPSULES TO ANIMALS

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During the conduct of experimental work on animals, it is sometimes desirable to administer by mouth a drug which is so unpalatable that it is difficult to induce the animal to take it, even though an attempt is made to disguise it in the food. The difficulty is increased if one wishes to give the drug without combining it with food. If the medicine is freely soluble, it can be administered by a tube passed into the stomach, provided that the solvent does not interfere with the experiment. Some drugs, however, are practically insoluble in the ordinary solvents, and administration of the latter group presents special problems.

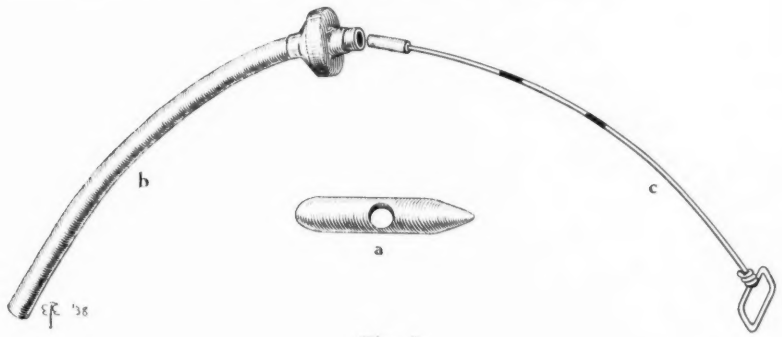

Fig. 1.

In order to overcome these difficulties, a simple instrument has been devised and constructed from very inexpensive materials. We have used this device for the administration of drugs to dogs, and probably its use could be extended to other laboratory animals. Its construction is somewhat similar to that of the balling gun, but it has certain distinct advantages over the latter instrument. It permits the administration of capsules of different sizes and the injection of several capsules at one time. Placing the capsules directly into the pharynx with a forceps or with the fingers is not only more difficult than the administration of drugs by means of our instrument, but it is also more hazardous.

The purpose of the instrument is to place the capsule or capsules into the lower pharynx, after which the animal swallows them involuntarily. It consists of a piece of moderately stiff rubber tubing of sufficient length to reach the pharynx (*b*). The tubing should have an inner

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diameter slightly larger than the diameter of the largest capsule to be injected and should be slightly arched in order to facilitate its introduction. To guard against the animal's swallowing the tube, a cork disk is placed around the proximal end. To prevent the animal from compressing the tube, a simple gag is used (*a*). When the tube has been introduced, the capsules are placed into it and are forced through it by means of a plunger (*c*) which is slightly longer than the tubing. The plunger is constructed from a piece of stiff wire, the end of which is made to fit the tubing by winding it with one-inch adhesive tape. The other end of the wire is bent to form a handle. Several riders, thin strips of adhesive tape wound around the wire at intervals between the handle and the opposite end, make the plunger more easily movable. The interior of the tube should always be kept dry and lubricated with powder.

This simple instrument, which can be constructed from a piece of broomstick (*a*), a short rubber tube (*b*), a cork, a piece of wire (*c*), and some adhesive tape, has proved very helpful to us in some of our work. We have administered daily for several weeks as many as twelve five-gr. capsules of cincophen and bile salts at one time.

CARCINOMA OF THE THIRD SEGMENT OF DUODENUM

Report of a Case

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CARCINOMA of the duodenum is a relatively rare condition. Compiled autopsy statistics totaling 350,286 cases reported by Eger[1] show the incidence of duodenal carcinoma to be 0.033 per cent. Hoffman and Pack[2] state that cancer of the duodenum constitutes 0.3 per cent of all intestinal cancer. In regard to the anatomic location, they report that 24.9 per cent are supra-ampullary; 61.9 per cent, peri-ampullary; and 13.0 per cent, infra-ampullary. Excellent reviews of this condition have been reported by Eusterman, Berkman, and Swan,[4] Lisa, Levine, and Fitzhugh,[5] and Mateer and Hartman.[6] No attempt will be made to summarize the literature since this has been done very ably by Meyer and Rosenberg[3] and Eger.[1]

Because of the rarity of cases involving the infra-ampullary or third portion of the duodenum and also because of the paucity of cases treated surgically, we wish to report a case which we have had the opportunity of treating.

CASE REPORT

The patient, a 47-year-old male, had been in good health until the spring of 1936, when he became ill for a few weeks, suffering from frontal headache, sore throat, nasal discharge, fever, and general malaise. Following this illness he did not quite regain his usual good health. In June of the same year he began having symptoms referable to his gastrointestinal tract; namely, loss of appetite, gaseous eructation, feeling of fullness after meals, and general epigastric distress. On several occasions he had a griping sensation in the epigastrium. He continued his usual occupation as a tool maker and in addition did some work in his garden at home. There was no diarrhea, constipation, tarry or bloody stools. In December the patient had an attack characterized by nausea and vomiting with abdominal cramps. In addition there was substernal pain, dyspnea, and weakness of the legs. A physician was consulted who prescribed iron which improved the symptoms. A few weeks later the vomiting recurred and the patient was admitted to a hospital where a gastrointestinal series was done. This was reported to be negative. The patient continued to have attacks of vomiting and upper abdominal distress. During his present illness, there was a weight loss of approximately 30 pounds.

Examination at the time of admission to the New Haven Hospital on June 14, 1937, revealed a moderately well developed male who did not appear acutely ill. Temperature and pulse and respiration rates were normal. The blood pressure was 120 systolic and 76 diastolic. The significant physical findings were limited to the abdomen, which was moderately full. The liver was felt one fingerbreadth

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below the right costal margin. In the midepigastrium there was an indefinite small mass which was slightly tender. There were no other masses or areas of tenderness. The laboratory findings showed a red blood count of 3,400,000 with 71 per cent Hb. (11 gm.) and a white blood count of 5,400. The differential count was normal. The urine showed a ± sugar and many mucous shreds. The stool was clay-colored and gave a 4+ guaiac reaction. Examination of the vomitus was negative for blood.

On the day following admission a gastrointestinal series showed almost complete obstruction of the third segment of the duodenum with considerable dilatation of the proximal duodenum and stomach (Figs. 1 and 2). Fluoroscopically, hyperperistalsis was noted in the stomach and duodenum. There was reverse peristalsis in the duodenum. At the site of the duodenal obstruction there appeared to be a mass which produced a pressure defect on the greater curvature of the pyloric antrum. The distal 6 cm. of the duodenum were irregularly narrowed and allowed the passage of only a small amount of the barium suspension. The duodenojejunal junction occupied a low position.

The x-ray diagnosis was obstruction of the third segment of duodenum; (?) carcinoma of pancreas infiltrating the duodenum.

The gastrointestinal series done at another hospital six months previously was reviewed. Unfortunately the duodenum was not visualized. There was no evidence of obstruction.

On June 19, 1937, an exploratory laparotomy was done. A firm intrinsic tumor in the region of the third portion of the duodenum extending from the duodenojejunal junction medially for a distance of about 5 cm. was palpated. There was no evidence of metastases in the liver or elsewhere. The tumor was movable laterally and was judged to be operable. The duodenum was not particularly distended. As a first stage, with the purpose of relieving the obstruction, a posterior gastroenterostomy was done. A relatively short loop of jejunum was used. The anastomosis was done by the semiclosed method, using two layers of interrupted silk sutures. The patient stood the operative procedure very well and his immediate postoperative convalescence was good. His intake of fluid by mouth was adequate and there was no vomiting. Stool examination showed a dark brown color with repeated 4+ guaiac reaction. On the seventh postoperative day, however, the patient vomited. Subsequently he vomited relatively large amounts once or twice a day, the vomitus containing large amounts of bile. Sufficient parenteral fluids were given to maintain an adequate water balance and he remained in fairly good general condition.

On July 8, 1937, the second operation was done under cyclopropane anesthesia with added small amounts of ether vapor. The previous operative incision was reopened. The jejunum was picked up at the ligament of Treitz and dissection of the third portion of the duodenum was begun and carried medialward, the duodenum being dissected free from the superior mesenteric vessels anteriorly and the aorta and vena cava posteriorly. After this dissection had been well begun, it was determined that the tumor had penetrated the posterior duodenal wall and invaded the retroperitoneal tissue. The transverse duodenum was dissected free, working through an incision in the transverse mesocolon. The jejunum was transected near its proximal end. The distal duodenum and proximal jejunum were then pulled through the space between the superior mesenteric vessels and the aorta. On further dissection of the transverse duodenum, it was found that the tumor extended along the posterior wall in the submucosal layer to within a few millimeters of the ampulla of Vater. Since it was impossible to close the duodenum distal to the common duct, this precluded the possibility of doing a duodenojejunostomy, which originally had been planned. The only alternative was a com-

plete duodenal resection. Accordingly the proximal duodenum was dissected free, transected between clamps, and the pyloric portion closed with interrupted silk sutures. The duodenum was then dissected free from the pancreas, the common bile duct being ligated at its junction with the bowel. The main pancreatic duct was not identified. However, the pancreatic tissue attached to the duodenum was ligated with silk ligatures and the entire duodenum was removed. The afferent loop of jejunum extending to the gastroenterostomy was resected proximal to the anastomosis and closed with interrupted silk sutures. A cholecystogastrostomy was then done, suturing the gall bladder to the adjacent anterior portion of the stomach, using a double layer of interrupted silk sutures. The rent in the mesocolon was

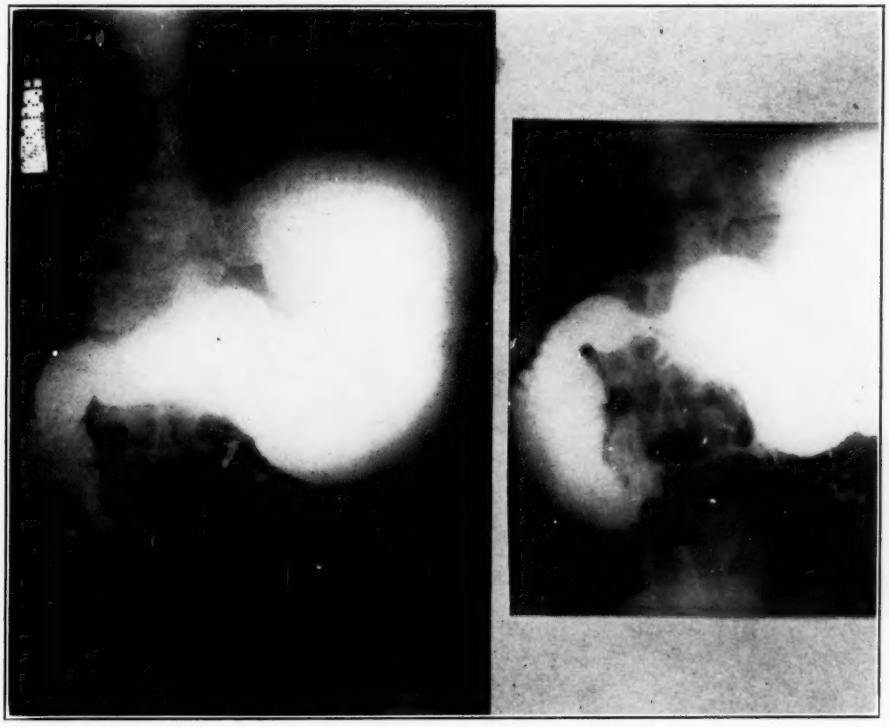

Fig. 1. Fig. 2.

Fig. 1.—Showing gastric and duodenal dilatation, and deformed, narrow, distal duodenum.

Fig. 2.—Six-hour film, showing gastric retention and dilatation of duodenum. Note irregularly narrowed third segment of duodenum and pressure defect on greater curvature of pyloric antrum.

closed with a continuous suture of fine silk. The wound was then closed in layers in the usual manner. At the close of this extensive procedure, which had lasted approximately three hours, the blood pressure had fallen somewhat despite an infusion which had been started a short time before completion of the operation. A 500 c.c. blood transfusion was given and the patient responded well. A few hours later, the blood pressure was again low and the pulse rapid. A second infusion was given which temporarily improved his condition. Despite two subsequent blood transfusions of approximately 250 c.c. each and additional parenteral fluids, the patient went into deepening shock and expired about eighteen hours following the close of the operation.

A post-mortem examination was done which showed extension of the duodenal carcinoma into the retroperitoneal tissue. Other autopsy findings were: fibrinopurulent peritonitis, hemorrhage in tail of pancreas, fat necrosis in mesentery, autolysis and gas bubbles in viscera (*B. welchii* septicemia), necrosis of muscles of anterior abdominal wall, bilateral hydrothorax. There was no evidence of leakage from the closed bowel ends or the cholecystogastrostomy.

Fig. 3.

Fig. 4.

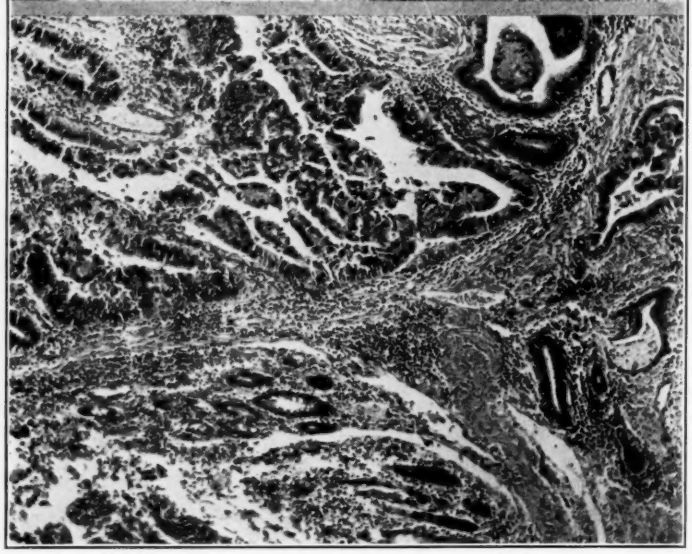

Fig. 3.—Photomicrograph showing transition from normal duodenal mucosa to disorderly tumor structure.

Fig. 4.—Photomicrograph showing cellular structure of the tumor. Normal mucosa seen in the lower left corner.

Histologic examination of the surgical specimen revealed replacement of normal mucosa by acinar structures lined by tall columnar epithelium, the nuclei of which varied greatly in size, shape, and staining properties. The pathologic diagnosis was adenocarcinoma of the duodenum (Figs. 3 and 4).

DISCUSSION

A preoperative diagnosis of carcinoma of the duodenum rarely has been made. Roentgenographic examination of the stomach and duodenum in recent years has been the means of establishing the diagnosis in a small but increasing number of cases. In the presence of a small tumor the examination may be entirely negative. If the tumor is more extensive, deformity and some degree of obstruction with dilatation of the stomach and duodenum proximal to the lesion may be seen. Carcinoma of the first portion may be indistinguishable from the deformity of chronic ulcer. If the tumor is bulky, it may produce a pressure defect on the greater curvature of the distal portion of the stomach. Such cases are difficult to distinguish from bulky carcinomas of the pancreas. The condition must be differentiated from tumor of the adjacent pancreas or biliary ducts, benign tumor of the duodenum, chronic duodenal ulcer, and diverticulosis of the duodenum.

In our case the symptoms and physical findings were indefinite and the exact location of the lesion was first established by the gastrointestinal series. The x-ray diagnosis was a tumor infiltrating the third duodenum producing a partial obstruction. That this was a tumor arising from the pancreas and secondarily involving the duodenum was believed to be most probable.

The ideal therapeutic procedure is resection of the involved duodenum and a duodenojejunostomy. In cases of congenital atresia of the third portion of the duodenum, duodenojejunostomy has been found to be the most satisfactory means of relieving the obstruction. Gastrojejunostomy does not adequately relieve distal duodenal obstruction since bile and pancreatic secretions accumulate in the proximal duodenum with regurgitation into the stomach followed by nausea and vomiting. In our case the gastroenterostomy did not relieve the patient's symptoms since he continued to vomit large amounts of bile-stained material. At the second operation it was planned to do a duodenojejunostomy following resection of the tumor and to unhook the gastrojejunostomy. Since it was necessary to resect the entire duodenum, the gastrojejunostomy was left intact and a cholecystogastrostomy was done. In the cases of carcinoma of the pancreas reported by Whipple and co-workers,[7] the pancreatic ducts were ligated in the course of resection and no immediate ill effects referable to the absence of the external pancreatic secretions were noted. From the recently reported experimental work of Ivy,[8] it appears that in dogs with pancreatic duct ligation survival for any length of time in most instances was possible only by feeding special diets containing large amounts of raw pancreas. This raises the question of the necessity of special dietary requirements and the prognosis of patients in whom the external secretion of the pancreas is absent. Our case offers no information in this regard.

SUMMARY

A case of carcinoma of the third portion of the duodenum with x-ray findings, surgical treatment, and post-mortem examination is reported.

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TRAUMATIC FAT NECROSIS

Report of a Case Involving the Buttock

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THE IMPORTANCE of recognizing traumatic fat necrosis rests upon the fact that clinically it resembles grossly carcinoma when the lesion appears in the breast, or sarcoma when the lesion appears elsewhere in the body. The condition appears sufficiently uncommon to justify the description of another case in a region in which, as far as could be determined, it has not been reported before, namely, the buttock.

CASE REPORT

S. S., aged 42 years, business executive, was first observed in consultation Nov. 4, 1936, at which time he presented ecchymotic areas over the left thigh and buttock, superficial abrasions of the left leg, and a swollen left knee. He gave a history of having been in a train wreck near Princeton, N. J. on Oct. 29, 1936, while traveling by rail from New York to Philadelphia. He and a number of other injured passengers were taken to the Princeton Hospital. After receiving first aid, he was discharged from the hospital and subsequently treated by his family physician. The patient was not again seen until June 8, 1937, seven months later, at which time he presented a hard painless mass in the left buttock, a condition which was not present at the time of the first examination. Believing that this mass was an organized hematoma resulting from the injury sustained in October, 1936, excision was advised. X-ray examination of the pelvis, including the hip joints, failed to reveal any bone involvement. The mass was clearly visualized in the soft tissues of the buttock. On July 1, 1937, the patient entered the Mount Sinai Hospital, of Philadelphia, where a more detailed history was obtained. He stated then that he had first become aware of an abnormal sensation in the buttock about six or seven weeks after the accident. He had noticed a stiffness of the left hip and some discomfort while sitting. Walking up stairs or changing positions from sitting to standing was difficult. He had some discomfort in crossing the left leg over to the opposite side. Bending forward was limited and painful. The patient interpreted these symptoms as manifestations of arthritis, and he consulted several physicians who apparently concurred with his views. In April, 1937, he visited Mount Clemens, where he took a "cure" for three weeks, but this made him worse. Upon his return to Philadelphia, he again sought medical advice and treatment, without very much benefit.

Finally, in the latter part of June, he decided to enter the hospital for operation. His previous medical history is unimportant. The patient is married and has two children. His habits are good. The family history is unimportant and irrelevant.

Physical Examination.—The patient is a short stocky man, with an excellent physique and good muscular development. The eyes, ears, nose, and throat are normal. The teeth are in excellent condition. The chest is well formed and resonant

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throughout; breathing is of the vesicular type. The heart is normal in size, shape, and position. The heart sounds are of good quality; no murmurs are audible. The liver and spleen are not palpable; no masses or hernial protrusions are present. The spine and extremities are normal, except for the left buttock, which presents a hard, painless tumor in the subcutaneous layers, globular in contour, and about the size of a tennis ball. Its edges are well defined. The skin overlying the mass shows no visible retraction or orange peel appearance. The mass is not raised above the skin, but appears to be fixed to the underlying structures. Flexion, abduction, and internal rotation of the left hip joint are restricted. Other movements are free.

Blood pressure, 120/72. Blood count: Hb., 94; RBC, 4,900,000; WBC, 6,200; Pmn, 65; SM, 33; LMN, 1; blood urea nitrogen, 14 mg. per 100 c.c. blood; blood

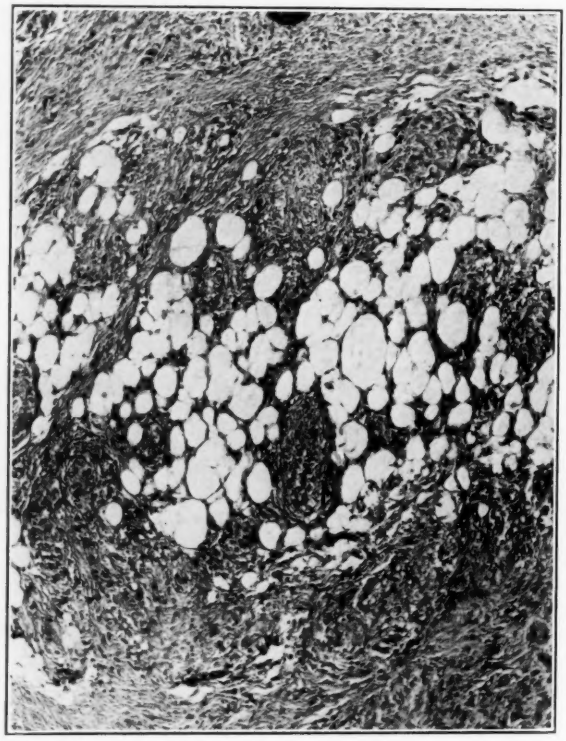

Fig. 1.—Subcutaneous tissue replaced diffusely by fibrous tissue and focally by tubercle-like bodies (×100).

sugar, 106 mg. per 100 c.c. blood; serology, negative. Urine analysis showed no abnormal findings. The preoperative diagnosis was organized blood clot or fibrosarcoma. At operation on July 2, 1937, under general anesthesia a five-inch oblique incision was made over the left buttock, centering over the mass. On dividing the skin and retracting gluteal fat, a rather startling condition was brought to view. The entire exposed area was infiltrated by a dense, grayish white fibrous tissue mass, extending into the gluteal fat, aponeurosis, and gluteal muscles. The mass was hard, globular, and sessile; the edges confluent with the dense fibrous fascial covering of the gluteus medius and maximus. The consistency of the tissue was that of cartilage, cutting with difficulty. Copious bleeding was encountered, but was controlled by hot, wet packs. The appearance of the lesion was strongly suggestive of a highly malignant fibrosarcoma. A frozen section revealed chronic

granulomatous tissue. No attempt was made to extirpate completely the mass since it would have entailed a sacrifice of muscle tissue. About one-half of the tumor was excised; the wound was coaptated with silkworm sutures, and a small rubber tube inserted for drainage.

Pathologic Report.—Macroscopic: The specimen consisted of two large pieces of tissue measuring 7 by 5 cm. and 6 by 3 cm. respectively. Some portions consisted of muscle, infiltrated, and replaced by a firm, grayish connective tissue. In other regions the original structure had been obscured by a firm gray tissue.

Microscopic: The subcutaneous tissue and voluntary muscle of the specimen had been extensively replaced by a diffuse and focal granulomatous-like process (Fig. 1). Many tubercle-like bodies were present composed of connective tissue

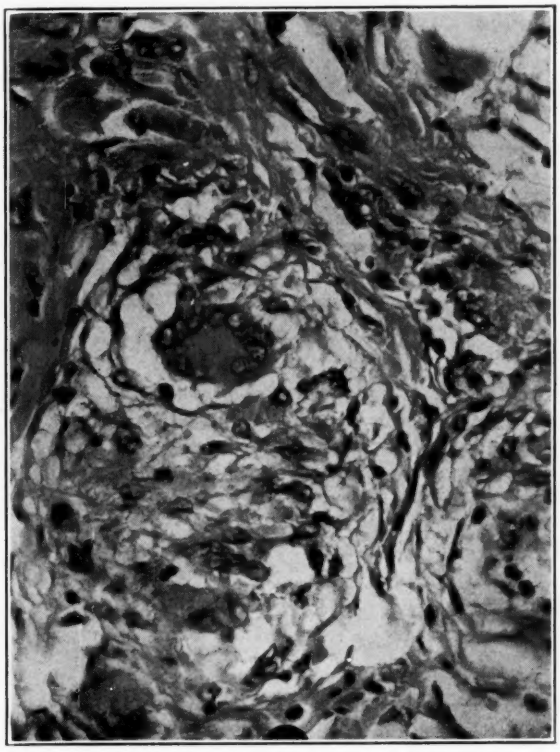

Fig. 2.—High power field of tubercle-like body showing giant cell (×600).

cells and epitheloid cells irregularly over-run with, or surrounded by, zones of round cells. The necrosis present in a few areas was not tuberculous in appearance. In some of the foci giant cells were present. (Fig. 2.) Between these focal lesions was an actively proliferating connective tissue process also over-run with lymphocytes and to a lesser extent with eosinophiles. In these areas giant cells were also present, unrelated in position to the tubercle-like bodies described above. Within nearly each tubercle-like structure and in the areas of diffuse involvement there were present fat cells. Some of the cells in the focal and diffuse lesions were phagocytosing fat. In the muscle the process was also present in association with collections of fat tissue located among the fibers (Fig. 3).

The slides were examined by Dr. Baxter Crawford, of the Jefferson Hospital, Philadelphia, Dr. Joseph McFarland, of the University of Pennsylvania Medical School, Dr. Charles Geschickter, of Johns Hopkins Hospital, Dr. Fred W. Stewart,

of the Memorial Hospital of New York, and Dr. R. H. Jaffe, Cook County Hospital, Chicago. These pathologists were unanimous in the opinion that the lesion was a benign inflammatory process. Dr. Stewart expressed the following opinion: "I find no evidence of tumor. The process is an inflammatory process, producing muscle degeneration, and subcutaneous and intramuscular fat necrosis, with a pseudo-tuberculous foreign body granulomatous reaction. Such processes, in fact, may be infectious or toxic. Since the break-down products of fat are irritants, even though infection subsides, the reaction may continue." Dr. Jaffe rendered the following opinion: "Foreign body granuloma with formation of numerous tubercle-like nodules composed of epithelioid cells and giant cells. According to my opinion, these nodules have formed around disintegrated tissue, and are secondary to fat necrosis. I did not see any histologic evidences of tuberculosis."

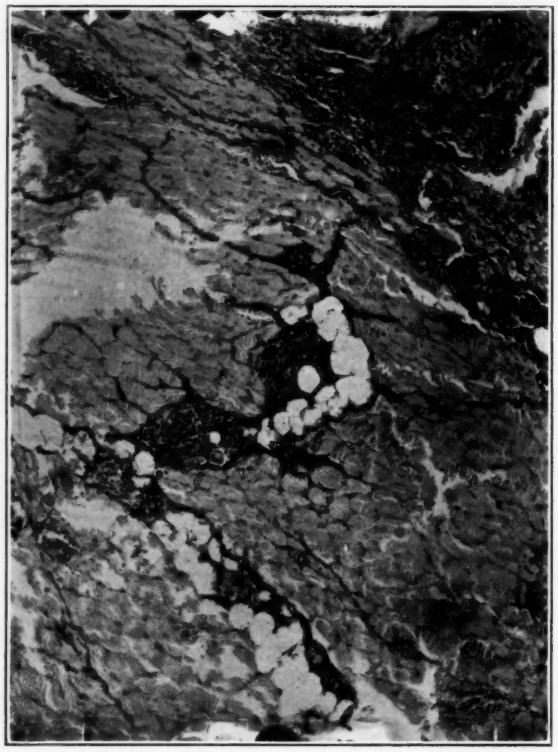

Fig. 3.—Showing involvement of muscle tissue by diffuse and focal processes (×100).

Clinical Course.—The patient remained in the hospital for ten days. His temperature rose to 101° F., and pulse rate to 130 per minute, following operation, gradually declining to normal in six days. Believing that x-ray therapy might be helpful in hastening resolution of the remaining portion of the lesion, the left pelvic region was irradiated on two occasions, July 7 and 9 respectively. Sutures were removed on the tenth day; wound healing was satisfactory, but a small sinus at the lower pole of the incision was still present.

Although reassured that the condition was not a sarcoma, the patient was greatly concerned about the outcome. He became despondent, anxious, and desired further consultations. The possibility of a tuberculous lesion worried him considerably. Accordingly, an x-ray of the chest was taken. It failed to reveal any evidence of tuberculosis. Direct smear studies from the discharge of the sinus,

which developed following the operation, were negative for tuberculosis. A guinea pig inoculation was performed and the animal was killed. No signs of tuberculosis were found. The sinus in the buttock continued to drain. The patient continued to worry and lost weight. The lymph nodes in the left inguinal region became enlarged and tender. In the latter part of July, the patient went to Rochester, Minn., where he was examined by Dr. Melvin Henderson, who advised exploration and further excision of the growth. On Aug. 6, 1937, the patient was readmitted to the Mount Sinai Hospital because of persistent pain in the buttock and drainage from the operative wound. The wound was laid wide open, more of the fibrous tissue removed, and the cavity was then packed with iodoform gauze. The packing was removed from time to time and the cavity irrigated with saline. At the end of about a month, healing was effected by a filling in of the cavity with healthy granulation tissue. With improvement of the local condition, the patient's general condition also improved. At the present time, March 1, 1938, he is in excellent state of health and has gained considerably in weight. There is no vestige of the mass in the buttock. There is, however, a defect in contour due to removal of gluteal fat, skin retraction due to the scar, and limitation of flexion and adduction of the hip due to the residual interstitial fibromyositis of the gluteus maximus and medius muscles. Because of this condition, the patient has some difficulty in operating his automobile and has some slight discomfort and stiffness when he sits too long in one position or on walking for any distance.

DISCUSSION

From a clinical standpoint, interest in traumatic fat necrosis seems to center about the lesion as it affects the breast. In this region the appearance of a hard, painless lump always excites a suspicion of carcinoma. This belief is fortified when skin retraction and fixation to underlying structures are present. Lee and Adair[1] in May, 1920, were the first to describe traumatic fat necrosis of the breast as a new clinical entity and to point out the clinical and pathologic differences between this benign and inflammatory lesion and carcinoma. Two cases were reported. In 1924 Lee and Adair[2] made a report on eighteen additional cases. C. E. Farr[3] in 1923 published a paper on "Ischemic Fat Necrosis" and demonstrated that the condition can be reproduced in guinea pigs by pinching or traumatizing the skin. These observations were recently confirmed by Proserpio[4] by a series of experiments upon laboratory animals. Farr reported seven cases occurring in regions other than the breast of similar etiology and with similar pathologic findings. He records a case of traumatic fat necrosis in a woman 30 years of age who six weeks previously sustained a severe contusion of the thigh. Examination revealed a hard, sausage-shaped mass, attached to the skin but not to the fascia lata. It was excised and pronounced sarcoma by a competent pathologist. Further examination proved it to be a case of fat necrosis, with typical giant cells and fat crystals. Production of fatty and fibrous tissue cells was quite pronounced. Farr[3] further reports cases in which hard lumps appeared in the subcutaneous fat, secondary to hypodermic injections, hypodermoclysis, and along an old healed suture line. W. B. Parsons[5] in 1924 reported a case of trau-

matic fat necrosis occurring in the groin of a boy 11 years of age, following a fall. Examination twelve days after onset of the injury revealed a firm, rectangular mass, 9 by 3 cm., attached to the skin, and readily movable on the deep structures. A section showed numerous giant cells of the foreign body type, round cells, fibroblasts, and large cells resembling macrophages.

In newborn infants the trauma of a difficult instrument delivery may produce a tumor having the identical microscopic picture of traumatic fat necrosis of the breast. De Vel and Bolin[6] in January, 1929, reported a case of a male infant, 21 days old, who showed several indurations involving the neck, left mastoid, and scapular regions. These lesions were attributed to the application of low forceps. When 6 weeks of age, biopsy of one of these indurations revealed increased amount of fibrous tissue, giant cells, normal lobulation of fat, and cholesterin crystals. When approximately three months of age, all the lesions had disappeared without leaving a trace. Traumatic fat necrosis in infancy has been adequately described by De Vel and Bolin[6] and more recently by Lacomme and Kreis-de Mayo[7] in 1936. In a review of the literature the important contributions on traumatic fat necrosis are by Lee and Adair,[1] Proserpio,[4] Cookson,[8] Hadfield,[9] Metge,[10] and Menville.[11]

PATHOGENESIS

Traumatic fat necrosis may be defined as a disintegration of fat cells due to trauma with associated tissue reaction, connective tissue formation, round cell infiltration, and a production of giant cells. The condition is less severe than the acute necrosis of fat associated with acute pancreatitis, in which relatively large areas become necrotic and cholesterin crystals are frequently seen. The factor of trauma as an exciting cause of the lesion is of considerable interest. When we consider the thousands of instances of injury to the subcutaneous fat and the comparative rarity of the reported cases of this condition, we must confess our ignorance of the mechanism which brings about the described changes in some individuals and not in others. Menville[11] believes the underlying cause to be a simple ischemia, produced either by trauma or pressure. The necrosis of fat is accompanied by a splitting into fatty acids and glycerin and is closely followed by repair. The presence of fatty acids in the tissues acts as a foreign body and sets up a chronic inflammation similar to that which occurs after injection of paraffin.

Hadfield[9] firmly believes that it is produced by sterile autoylsis. Proserpio[4] would call this partial splitting into the fatty acids and glycerin of the neutral fats of the subcutaneous tissue, subcutaneous lipo-necrosis, and believed that the process should be regarded solely as an abnormal reaction of the connective tissue to this modification of fatty tissue, which he believed is provoked by trauma in a subject in whom the powers of defense are altered or weakened.

CLINICAL CONSIDERATIONS

In describing the clinical features of traumatic fat necrosis, cases occurring in newborn infants and in breasts will be excluded, and the remarks confined to lesions occurring in adults. A history of injury is usually obtained. A hard lump appears at the site of injury which continues to increase in size but is not painful unless pressure is made by the mass upon adjacent nerves. If it occurs at, or near, a joint, there will be limitation of movement and stiffness if the underlying structures are involved. The mass is fairly well circumscribed, extremely hard, fixed to the skin and occasionally to the deeper structures. The tumor may appear in from a few days to ten years after the trauma. It varies in size from a small discrete nodule with well-defined sharp edges to the size of the palm of a hand or even larger. There is very little elevation above the skin. The skin may or may not be retracted or puckered. In early cases the skin may be reddish or purplish. When the lesion occurs in the buttock, the mass may assume tremendous proportions. The skin over the buttock is a relatively thick structure, loosely connected with a superficial fascia which is abundant, and contains a large quantity of poorly organized fat. The subcutaneous layer here is poorly supplied with blood as compared with other subcutaneous areas. The skin is coarse, with numerous sebaceous follicles, and is frequently the site of minor forms of irritation and bruises. The laxity of the superficial fascia permits effusion of pus or blood and fat necrosis to attain exceptionally large dimensions. Based upon the above anatomic considerations, the gluteal fat must be naturally predisposed to the development of the lesion, called "lipo-necrosis." It is highly probable that the small hard discrete nodules or lumps that are frequently observed in patients who have received subcutaneous or deep intramuscular injections, if examined microscopically, would reveal the histologic picture of fat necrosis. These lumps are innocuous and painless; hence they go unnoticed, or are ignored by patient and physician. A similar lump appearing in the breast, however, is usually treated with greater respect, for the fear of malignancy prompts investigation. Local excision, even when partial, will suffice for adequate treatment. Regeneration begins after excision and soon no trace of the original mass remains.

PATHOLOGIC FEATURES

Grossly the affected region is pale, opaque, dull, grayish white, hard, and homogenous. In our case, fat, aponeurosis, and muscle were involved. Muscle is rarely involved. Microscopically the characteristic findings are collections of small round cells often containing free fat (microphages), large mononuclear (macrophages), and multinuclear cells (giant cells), also often fat laden, and frequently interstitial fatty acid crystals in association with a spindle cell stroma of varying

density. The presence of fatty acid crystals is not essential for the diagnosis of fat necrosis. In our case the striking features were the large number of pseudotubercles.

According to Menville,[11] subcutaneous fat necrosis is represented by two distinct phases, degeneration and regeneration. Microscopically, the degenerative phase is characterized by the presence of microphages and macrophages containing degenerating fat in counterdistinction to the regenerative phase which is characterized by the invasion with connective tissue elements. Both phases are seen in nearly all cases of fat necrosis; the predominance of one type of reaction over the other is the deciding factor in determining the prevailing stage of the process.

CONCLUSIONS

A case of traumatic fat necrosis of the buttock is placed on record. Clinically, the condition was characterized by the presence of a hard, tumor mass in the subcutaneous layers of the buttock. Attention was drawn to the gross resemblance of the lesion to sarcoma. Its true nature was determined by microscopic examination, which revealed necrosis of subcutaneous and intramuscular fat, giant cells containing fat, round cell infiltration, pseudotubercles, and excessive fibrous tissue formation. Partial excision resulted in a cure.

It is believed that the pathologic entity known as traumatic fat necrosis represents a cellular response of the subcutaneous tissue to fat metabolites. Local trauma and ischemia are capable of splitting fat into fatty acids and glycerin. These end products, it is believed, produce the characteristic histologic picture of this disease.

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CHRONIC HYPERTHYROIDISM*

II. Diffuse Toxic Goiter

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IN 1936, in collaboration with Krantz, a group of cases of chronic hyperthyroidism (nodular goiter) occurring in a series of 450 consecutive patients on whom thyroidectomy had been performed was reported. Since the publication of this study, other articles pertaining to this long neglected phase of hyperthyroidism have been published. Due to the interest of Davison, Hertzler, Plummer, Mayfield, and others in this much misunderstood and variously diagnosed group, there is beginning to be manifest an increasing care in differential diagnosis and subsequent advice and treatment. Search of available literature, however, has failed to reveal any articles in which results following thyroidectomy in diffuse toxic goiter characterized by chronicity have been studied and correlated with the symptoms existing and the pathology found.

To say the least, it takes the courage of a firm conviction to attempt to cull from the rather large field of cases usually diagnosed neurosis a series in which thyroidectomy is advised and carried out. Such an action means invasion of the field usually assigned to the neuropsychiatrist, the internist, or the general practitioner. In short, this group includes any individual who is diagnosed as a neurotic, a psychoneurotic, or as having a neurocirculatory asthenia, sympathicotonia, effort syndrome, autonomic imbalance, soldier's heart, cardiovascular neurosis, vasomotor neurosis, or any other function neurosis. It is not intimated that these conditions do not exist, but rather that some of these cases may and do fall into the group of diffuse goiter of the chronic type, or low grade, borderline hyperthyroidism.

Morris, Levine, and others dwell on another phase of chronic hyperthyroidism often diagnosed incorrectly, where the outstanding symptoms are cardiac and the patients are treated as such. Plummer believes that as practitioners we should study to become "goiter conscious" and "that in any case of failing health not obviously and entirely explained after careful examination, hyperthyroidism should be considered." In addition to the functional neuroses and cases incorrectly called heart disease, there are other conditions which may be confused with borderline or chronic hyperthyroidism. Early low grade essential hypertension may simulate it very closely. Also, gen-

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eral physical debility as related to overwork, improper nourishment, nervous strain, and a variety of other causes must be ruled out. Such is also the case with the asthenic individual constitutionally marked early in life as a substandard, high-strung type of person with a predisposition to nervous system imbalance. In the diagnosis of this disease, more than in any other involving the thyroid gland, one must tread cautiously and carefully. Classifying a case of borderline or chronic hyperthyroidism in a diffuse goiter as one of the functional neuroses and treating the condition as such will not alleviate the symptoms. As in the cases herewith reported, it will only induce the patient to seek help elsewhere until finally a correct diagnosis is

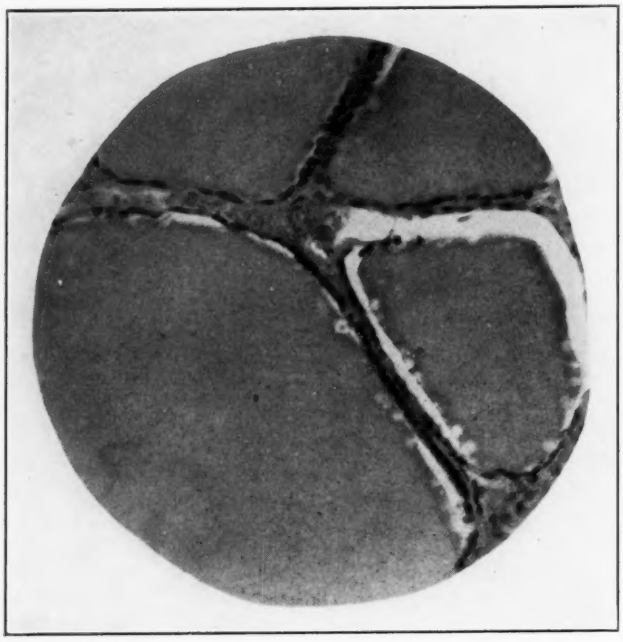

Fig. 1.—Typical diffuse goiter showing distended acini filled with colloid in which there is slight vacuolization. The epithelium is flat or low cuboidal.

arrived at and the proper treatment established. Conversely, subtotal thyroidectomy performed in cases of functional neurosis, essential hypertension, or myocardial degeneration will not cure or improve the patient. It may have the reverse effect, particularly in the neuroses, in that it aggravates the condition through the realization by the patient that he has been subjected to an unnecessary operation. On the other hand, it is equally as important not to operate upon this group, unless a diagnosis of hyperthyroidism is established, as it is to operate when such a diagnosis can be made with a reasonable degree of certainty. Jackson reports 100 cases where thyroidectomy previously had been advised elsewhere. In this series there was disagreement with the diagnosis and none of these cases was subjected

to such operation. A careful follow-up of these patients over a considerable period would be very interesting. It seems from the report alone that definite conclusions can hardly be accurately drawn.

We have progressed a great deal since the day when the feeling existed in medical circles that no conscientious surgeon would operate on goiters to the present time where we are actually operating and curing borderline cases heretofore considered as some type of functional neurosis. To advise thyroidectomy in these borderline cases, without adequate study, will most surely lead to a high percentage of error both ways; that is, in refusing operation to some who should have it and in advising operation for others where it is not indicated.

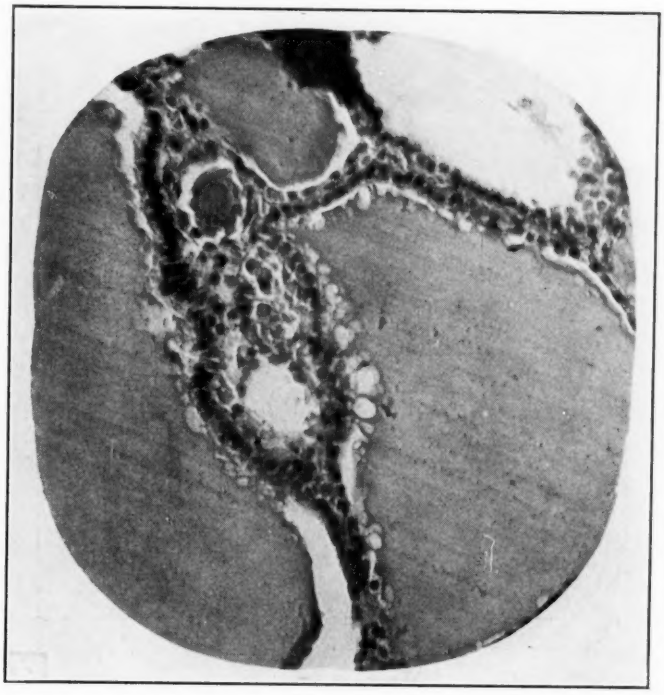

Fig. 2.—Diffuse goiter with beginning formation of columnar epithelium. There is also increased vacuolization of colloid.

Adequate study cannot be done without time for repeated clinical examinations, basal metabolic determinations, and other necessary laboratory procedures. Careful study and consideration should be augmented by the therapeutic iodine test. If the metabolism drops and the patient is clinically improved following a week to ten days on twenty to thirty drops of compound solution of iodine daily, it is fairly safe and reasonable to assume that hyperthyroidism exists. In the series herein reported, the average lapse of time between the original examination and thyroidectomy was thirty-nine days. During this period the patient was seen and studied a number of times,

metabolisms were often repeated, and the iodine test was used. Only then was an attempt made to draw conclusions with a reasonable degree of accuracy.

Much can be said of the inaccuracies of the metabolism test and of how misleading it can be if not properly interpreted. The machine itself is subject to error. Any leak or defect in the mechanism tends to a higher false reading. Technicians may err in their figures; the patient may have a fever, a cold, or inadequate rest the night before or just before taking the test. Not infrequently the patient is not in the postabsorptive or true basal state due to inadequate rest or ingestion of food. This, of course, will give an abnormally high energy

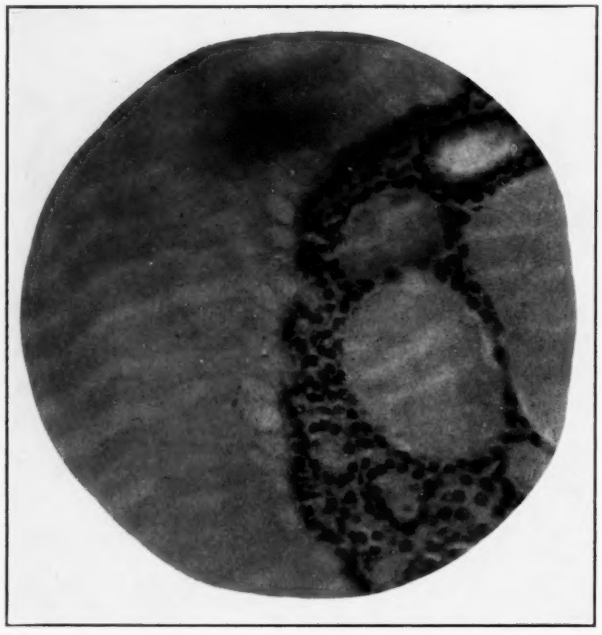

Fig. 3.—Diffuse goiter with early inbudding of the epithelium. There is also a tendency to form new smaller acini.

exchange. The result is merely a metabolism determination and not basal. Another fact perhaps not recognized generally is that 20 per cent of normal people have basal metabolisms outside the normal range of +10 to –10, and that a considerable number of them will fall between –10 and –20. For example, if an individual of this group shows clinical signs of mild hyperthyroidism and has a metabolism reading of 0 to +5, there is definite elevation of his basal rate.

In a study of any case where the metabolism is used as an adjunct to the clinical findings, the possibility of technical error must be considered. If the patient is somewhat frightened and stimulated by a type of examination which is wholly new to him, it is advisable to take two or even three metabolisms. Should the rate still remain

above normal, a drop of 10 to 15 points following the therapeutic iodine test may be a significant factor in diagnosis. A drop of +15 to +2 is no more significant, however, than a drop of +7 to −6.

Authorities are increasingly cognizant of the fact that a metabolism reading cannot be taken literally. Plummer frankly says "we must modify our increasing and immature faith" in a single reading. Davison believes it is inadvisable to accept the metabolism determination as proof of the presence or absence of hyperthyroidism. The metabolism must be considered as a valuable adjunct in the diagnosis of hyperthyroidism, but, where it is in disagreement with the clinical

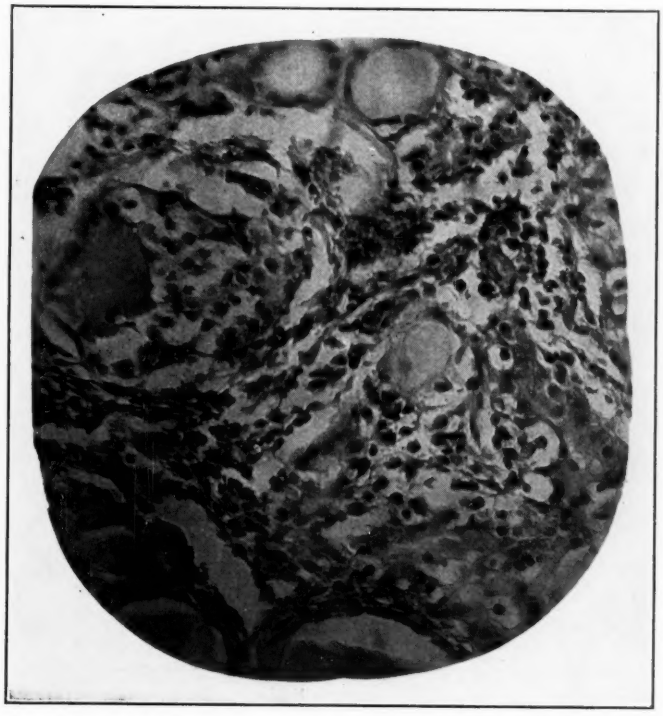

Fig. 4.—Diffuse goiter with moderate degree of lymphocytic infiltration and increased connective tissue formation.

picture, it should not be considered. This is more particularly true in the borderline cases than in the more severe types.

The differential diagnosis between chronic hyperthyroidism or mild low grade thyroid intoxication and the various functional neuroses must be done with great patience and care. First, an adequate, careful history must be taken, always searching both in the family history and the personal history for a clue which may possibly shed light as to why the complaints are suggestive of a neurosis. If such a definite clue exists, the chance of this being a low grade hyperthyroidism is decreased. Plummer stressed fear complexes as being responsible for exaggerated physiologic reactions. The subjective symptoms may

vary greatly in mild or borderline hyperthyroidism. Any or many of the usual symptoms of toxic diffuse goiter may be absent. Quite frequently the pulse may be normal or only slightly elevated. In some there is no loss of weight and occasionally there is a case in which a fairly recent moderate weight gain is present. Tachycardia is one of the most constant symptoms, but it may be absent. Where it exists, it is usually present with excitement or exertion or after eating. The pulse need not necessarily be increased all the time as in severe thyrotoxicosis. As a rule dyspnea is not present in a large number

Fig. 5.—Low power. Diffuse goiter with marked lymphocytic infiltration. Definite thyroiditis.

as severe cardiac damage has not been done as yet. Appetite may be increased, normal, or decreased, and is not constant enough to be significant.

The nervous symptoms which approximate those of the functional neuroses should be evaluated with great care. A high percentage of these people will have excitability, irritability, and emotional instability in some form or other. Tremor of the fine type is also present frequently. These people are not often depressed as is the psychoneurotic. The hands and feet are usually warm and moist in contrast to the true neurotic who complains of always having cold hands and feet. Loss of strength and lack of endurance are quite constant symptoms. The desire and energy to do things is usually

present without the physical ability to carry them out. This is in contrast to the neurotic who may be tired all the time and utterly lacking in ambition. Careful questioning as to the amount of clothes worn and as to bedclothes used both in winter and summer will indicate whether or not heat intolerance is present. It is by no means a constant symptom, but is found in a reasonable percentage of cases.

Generally speaking, objectively we have an individual, usually in early middle life, slender, asthenic, intellectual, tense, easily exhausted. The blood pressure is elevated moderately with a relatively low diastolic level and an increased pulse pressure. The thyroid gland is usually moderately enlarged, firm, elastic, and frequently causing pressure symptoms. The basal metabolic rate is either normal or moderately elevated and in an occasional case may actually be subnormal according to average standards.

In a consideration of low grade hyperthyroidism in diffuse goiter, 500 consecutive cases operated upon by myself were studied and those cases included in this report which were considered borderline or questionable at the time of the first examination. However, no cases were included whose metabolism was over +30 and it is significant that none of the higher metabolisms ranging from +20 to +30 could be considered, at the time of the original examination, frank cases of hyperthyroidism. Considerable study and care were given to ruling out possible neurosis. Each case had a pathologically proved diagnosis of diffuse goiter after operation, and no case having even a small nodule was included. A considerable number of this group had two and some had three metabolism studies and in many a therapeutic iodine test was done before advising thyroidectomy. There were 55 cases included in this series or 11 per cent of the whole group. The highest metabolism before operation was +30 and the lowest −12, while the average preoperative metabolic rate was +15.6. The postoperative average taken from two to six months after operation was −0.8. This showed a drop of 16.4 points in the metabolism rate following thyroidectomy.

The oldest patient operated upon was 66 years old, the youngest 15, and the average age was 32 plus. There were 12 males and 43 females included. As stated before, no patient was advised to have thyroidectomy without having careful, adequate study and the average time elapsing between examination and operation was 39 days. The highest systolic blood pressure recorded was 180 and the lowest 96, while the highest diastolic was 110, and the lowest, 54. The average for the 55 cases was: systolic, 144; diastolic, 74; making a mean pulse pressure for the group of 70.

In considering the symptoms, both the office histories and the hospital records were studied and the findings correlated as nearly as possible. The average duration of symptoms was 14 months, the long-

est 8 years, and the shortest 2 months. In all cases, however, these patients dated the onset of their disturbance to a reasonably definite date. Thirty-four complained of weight loss, the greatest loss being 35 pounds, while 3 patients actually showed a gain in weight and 18 noted no change. The average loss for the 34 patients reporting it was 11.2 pounds. Twenty patients complained of choking, pressure or difficulty in swallowing at times.

The symptoms referable to the nervous system were most predominant. Fifty-four complained of nervousness; 49, increased excitability; and 41, irritability. Tremor was present in 38. Tachycardia was present and noticeable to the patient in 50, and dyspnea in 44. There was definite heat intolerance in 21, and increased appetite in 16, while 43 designated weakness or lack of endurance as one of their symptoms. This is an average picture of the symptoms complained of by the group as a whole. All of them had moderately enlarged glands varying from slightly above normal in size to a very definite, easily palpated bulging deformity of the neck. As stated previously, partial thyroidectomy according to established standard technique was done on this group of 55 individuals.

An effort was made to evaluate the benefits derived in these cases following thyroidectomy. Questionnaires were sent to all of these people and answers received. It was felt that sufficient time should elapse to insure full recovery and, with this in mind the, questionnaires were sent to patients from two to five years after operation. Many questions were included, chief among them referring to symptoms usually found in diffuse toxic goiter. The first was: "Were you entirely relieved of the symptoms of which you complained at examination, following your thyroidectomy?" Forty-two answered this question in the affirmative; 8 stated they had been relieved, but did not consider themselves entirely well as yet, while 5 answered definitely "no."

The average time which elapsed before the patient felt normal was 9.1 months. Sixteen patients stated they had some degree of tremor left; 16 were still excitable to a degree, while 15 still considered themselves more irritable than normal; 14 answered that they still had tachycardia at times and 7 that dyspnea was still present with exertion. Only 2 reported increased appetite and 1 heat intolerance; 44 stated that their weight was now normal and 11 that they were still underweight. Strength was reported as normal by 43, and subnormal by 12. The response to the questionnaires showed that 76.3 per cent of these people considered themselves cured, 14.5 per cent improved but not entirely free from trouble, and 9.2 per cent unimproved. There are two explanations for the 9.2 per cent unimproved. Referring to Chart

I, it is noted that following operation the last 4 patients still maintained a metabolism reading very definitely above normal. This would indicate that they had not been relieved by thyroidectomy and that either a recurrent or continuing hyperthyroidism existed. Further follow-up study of these 4 cases should be done. However, at this point the records are incomplete as the patients failed to return for further examination. As there were only 5 cases in the unimproved group, this would leave 1 case, or 1.8 per cent, which was undoubtedly a mistaken diagnosis and should not have been operated upon. Chart I shows further the drop in metabolism in each individual case following operation. In 6 patients, the metabolism was moderately

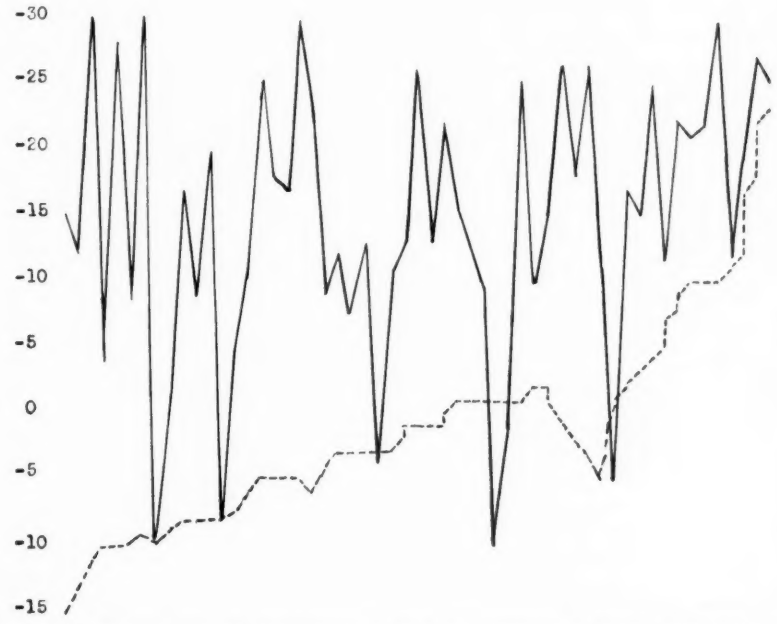

Chart I.—Comparison of preoperative and postoperative basal metabolic rates. Preoperative readings are shown by the continuous line; postoperative, by the dotted line.

higher after operation than before, though still within normal range.

As a means of further substantiating the contention that thyrotoxicosis existed in a majority of these cases, a study was made of the pathologic specimens removed at operation, to correlate if possible with the subjective symptoms and the results obtained. As would be expected with a group such as this, a relatively small percentage showed the typical gland of diffuse toxic goiter with marked increase in the cellular structure. It must also be kept in mind that all of these patients had Lugol's solution preoperatively which would, to some extent, effect the pathologic picture by a restoration of colloid and a decrease in the epithelial hyperplasia where it existed.

TABLE I

SUBJECTIVE SYMPTOMS

	PREOPERATIVE	POSTOPERATIVE
Loss of weight	34	11
Loss of strength	42	12
Choking; pressure in neck or difficulty in swallowing	20	0
Nervousness	54	15
Excitability	49	16
Irritability	43	15
Tachycardia	50	14
Dyspnea	44	7
Heat intolerance	21	1
Increased appetite	16	2

It is of interest to note the various pathologic diagnoses as made by a number of different pathologists on this group of cases. Table II shows 19 different diagnoses made on the group of 55 cases which clinically fell into the classification of diffuse toxic goiter (chronic). In this series the average weight and average measurements of the specimens removed were tabulated. These were found to be: weight, 42.8 gm., while the measurements were 11.1 by 7 by 3.8 cm., making an average bulk of 273.1. An effort was made to determine if a possible relationship existed between the different ranges of metabolism and the weight and bulk of the gland removed, and compared with an average normal gland. As is shown in Chart II, the average weight of a normal gland is 30 gm.; the bulk, 211.6. In the metabolism range of +20 to +30, the mean weight of the specimen removed was 37.5 gm., and the bulk, 280.1; +10 to +20, 51.5 gm. weight and 311.6 bulk; 0 to +10, 37 gm. weight and bulk 321.9, while in the 0 to –10 or below

TABLE II

VARIETY OF PATHOLOGIC DIAGNOSES ON A SERIES OF 55 CASES OF DIFFUSE TOXIC GOITER (CHRONIC)

Simple goiter
Simple goiter with epithelial hyperplasia which has undergone degeneration
Colloid goiter
Diffuse colloid goiter
Diffuse colloid struma
Cystic degenerating colloid goiter
Struma diffusa colloides
Struma diffusa microfollicularis
Thyroiditis
Chronic thyroiditis
Treated exophthalmic goiter with thyroiditis, Grade 2
Hyperplastic goiter
Mild diffuse thyroid hyperplasia
Hyperplastic thyroid following iodine
Struma diffusa with epithelial hyperplasia, Grade 2
Struma diffusa with epithelial hyperplasia, Grade 1
Early exophthalmic goiter
Hyperplastic goiter undergoing iodine degeneration
Exophthalmic goiter

group, the gland was slightly less in bulk but not in weight compared with the higher metabolism levels. This fact would not seem to have any particular significance.

In a microscopic study of the specimens removed, it was found that 14 of the 55 cases were diffuse goiter or simple colloid goiter. This accounted for 25.4 of the total. Referring to the answers to our questionnaire, it is noted that all but 23.7 per cent of the cases considered themselves cured. From the purely pathologic standpoint, it would be expected that those included in the 25.4 per cent might be considered doubt-

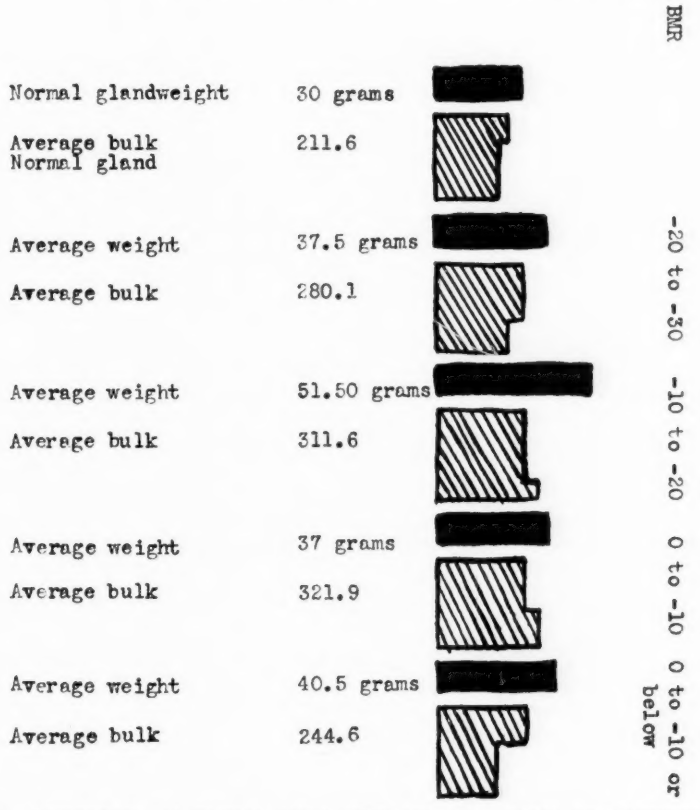

Chart II.—Comparison of normal gland with average weight and average bulk of thyroid tissue removed at different basal metabolic levels.

ful cases. From the standpoint of benefit obtained, the percentages then jibe very closely. This, however, does not account for the 14.5 per cent who did not consider themselves entirely cured but who had shown definite improvement. This might be explained by a possible psychologic benefit which might follow an operative procedure or by the fact that there are certain colloid changes not fully understood where thyroidectomy is of definite benefit. Seventeen cases were typical pictures of diffuse goiter with varying amounts of lymphocytic infiltration.

Twelve specimens were characterized by increase in the fibrous stroma from moderate to marked degree, and also by slightly thickened or columnar epithelium, in some cases showing degenerative tendencies.

Ten cases without doubt fall into the classification of diffuse toxic goiter as we are familiar with it. They show proliferation of the epithelium with slight to moderate inbudding and definite increase in the cellular structure.

Two cases showed marked lymphocytic infiltration and increase in the fibrous stroma and could be definitely classified as thyroiditis.

Hertzler emphasizes the importance of regressive changes in colloid structure plus lymphocytic infiltration and connective tissue changes as confirmatory evidence of thyroid pathology. In many cases of this type there is little doubt but that some degree of epithelial hyperplasia could be demonstrated had not the patient been treated with iodine over varying periods. If the gland removed shows no colloid regression or vacuolization, no interstitial increase or invasion with lymphocytes, and the epithelium no evidence of becoming columnar or beginning inbudding, it is reasonable to assume that the operation has been done probably on a case of functional neurosis and that little or no benefit will result. Conversely, if any or all of the above conditions obtain in the specimen removed, we may conclude that the operation is justified and that the subsequent cure of the patient will corroborate it.

CONCLUSION

The results of this study would indicate that a definite borderline group of diffuse toxic goiter exists, which is quite generally confused with various functional neuroses. Further, that it is primarily important to give these cases adequate study before classifying them either as diffuse toxic goiters or as functional neuroses. Finally, that such a policy will not only serve to relieve and help many people who are true borderline or chronic hyperthyroidism cases, but will also prevent unnecessary operation on those who do not fall into this group.

SUMMARY

Fifty-five cases of chronic hyperthyroidism in diffuse goiter have been considered from the standpoint of subjective findings and results after thyroidectomy and are correlated with a study of the pathologic specimens removed. Thyroidectomy has been responsible for cure in 76.2 per cent, improvement in 14.5 per cent, and no change in 9.2 per cent. The end-results justify thyroidectomy in carefully selected, thoroughly studied cases in this group. There was no mortality in this series.

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A PRELIMINARY REPORT ON THE USE OF ZINC PEROXIDE IN MOUTH INFECTIONS*

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THE work done by Meleney[1-4] and others showing the value of zinc peroxide in the eradication of infective processes caused by anaerobic bacteria, particularly in the case of deep burrowing ulcers, led us to investigate its action in the mouth. While our study was going on, Meleney specifically suggested its value in certain mouth cases.[5, 6]

It is well known that the organisms which do the greatest amount of damage to the gingival tissues are anaerobes, particularly those occurring in Vincent's disease. The success which has attended treatment of oral pathology caused by these bacteria in the past is attributable to the fact that normally anaerobic areas can be converted into aerobic ones by the introduction of pure oxygen. To effect a cure, in addition to necessary local treatment by the dentist or physician, it is imperative that the patient give supportive home care with oxygen-liberating compounds. Hydrogen peroxide and sodium perborate generally have been relied upon to furnish this aid. Both of these compounds, if used frequently enough over a long period of time, will produce enough oxygen to kill the anaerobic pathogens of gingival infections. On the basis of results already obtained and reported here, it is believed that the same effect can be accomplished with zinc peroxide in a shorter time, with less irritation to the tissues than usually accompanies treatment of such infections, and with several other important advantages.

Zinc peroxide (ZnO_2) is a white, amorphous powder, resembling zinc oxide in appearance. It is manufactured by the R. & H. Chemicals Department of E. I. duPont de Nemours & Company, Niagara Falls, N. Y., and is now available for clinical use. It is quite stable and comes to us not as a pure salt but contains: 45 to 50 per cent zinc peroxide, 35 to 40 per cent zinc hydroxide, and 10 to 15 per cent zinc carbonate.

Various batches behaved differently for Meleney when he first used the preparation. Those batches we have used were essentially the same and it is my understanding that the product is now definitely standardized. The "medicinal grade" only should be used and must be heated to 140° C. in quantities of not more than 500 gm., for 4 hours, to effect sterilization, in accordance with the manufacturer's specifications. It should be pointed out, in passing, that all zinc peroxide obtainable is not a "medicinal grade."

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To determine whether or not the preparation is effective, a simple test has been devised.[4] Place 1 dr. of the powder in a test tube and mix it with enough water to form a creamy suspension. Effective preparations sediment quickly, leaving a clear, supernatant liquid. Bubbles will appear in the tube in several hours and at the end of 24 hours the sediment is raised up by the gas bubbles in a curdy mass to several times its original height. Gas evolved is almost entirely oxygen. Ineffective preparations leave a milky supernatant liquid and produce little gas. In these ineffective preparations, the sediment may harden like plaster.

In the management of mouth infections involving the gingivae and periodontium, we have not changed from an opinion given some time ago,[7] that more than one medicament is necessary for the local treatment of these conditions. The organisms seem quickly to develop a tolerance for one drug. Hence, we have employed chromic acid, 8 per cent; acriviolet, 1 per cent; acriflavine, 1 per cent; gentian violet, 1 to 5 per cent; tincture of metaphen, merthiolate, and very occasionally sulpharsphenamine in glycerine suspension. In this connection, the work of Keilty,[8,9] in which he describes a technique for the selection of certain drugs for specific infections by impregnating cultural media with the medicaments, is pointed out. Frequently, only two or three of those drugs mentioned, used alternately, are required, but in persistent cases we have resorted to the use of all of them. It has been our experience that unless some rotation of treatment is employed the course of the recovery will be definitely delayed. We have used ultraviolet light (air-cooled) in increasing doses from ½ minute to 3 minutes per radiated area, with gratifying success, as an adjunct to our other medication for the past two years. And until we began the use of zinc peroxide, sodium perborate was our drug of choice for home treatment. It was prescribed in the mixture of a level teaspoonful to a half-glass of warm water.

The real effectiveness of sodium perborate depends upon placing it in solution and using that solution as a mouthwash. It contains 10 per cent of available oxygen but gives off that oxygen quickly once it is placed in solution. This characteristic is even more pronounced when the perborate comes in contact with organic substances, such as are found in the mouth. Hydrogen peroxide, in the usual 10 per cent solution used medically, has 1.3 per cent of available oxygen. It also loses its oxygen very rapidly and must be kept in an acid medium to retain its stability. Zinc peroxide, on the other hand, with 7.5 per cent of available oxygen, nearly as much as sodium perborate, loses its oxygen very, very slowly. Therein lies its outstanding virtue for the purpose to which we wish to adapt it.

It has been pointed out that hydrogen peroxide is kept stable by the addition of slight amounts of acid. As it is dispensed, the pH will be

between 1 and 2. The small amount of acid can easily be neutralized, however, but when this happens the compound gives up its oxygen at once. Sodium perborate, when placed in solution, will register a pH of 10.5 and this highly alkaline reaction is quite undesirable. It becomes very irritating to delicate membranes and particularly to new granulations when its use is prolonged. Zinc peroxide has a pH of 8.8 and remains very stable over a long period of time, giving up its oxygen so slowly that great amounts of it can be utilized to permeate interstices, crypts, and pockets where organisms associated with all types of gingivitis, pyorrhea, osteomyelitis, and soft-tissue necrosis abound. Further, the zinc oxide residue is bland and healing and this is an added desirable characteristic.

Zinc peroxide has been known for a long time to be a powerful deodorant and in a form not known as a "medicinal grade" has been used in many ways to combat offensive smells. Deodorization is a process of oxidation and it seems reasonable to believe that the ability of any given substance to bring about oxidation of another substance is directly proportional to the length of time the oxidizing substance can continue to give off oxygen. Hydrogen peroxide and sodium perborate, it has been shown, lose their oxygen supply quickly. Zinc peroxide does not do this and, therefore, will continue to react almost as long as it is possible to keep it in contact with tissue, particularly that of the oral cavity.

We have altered our method of treatment of gingival infections for the cases reported in only one respect—the substitution of zinc peroxide for sodium perborate. Without exception, the cases have responded better than they previously did. Perhaps the answer is not only in the fact that more oxygen is available for use from the zinc peroxide. It may be that the patients find it easier to use the zinc compound than the perborate. I do know that those who work in offices find it much simpler to apply the zinc at intervals of three or four hours than to use the sodium perborate or hydrogen peroxide every hour, as it was necessary to prescribe it in acute cases. In the two cases of necrosis following radiation, every available medicament had been tried without much success. In the cases of suppurating periodontoclasia almost the same had been true, although it must be said that some improvement had resulted from the use of sodium perborate.

It will be noted, in the case reports, that several methods of applying the zinc peroxide were used. After some experience with the compound, it was finally decided that our best results were obtained when it was applied as a paste.

It is not the purpose of this paper to discuss at length the various methods of local treatment of mouth infections after a diagnosis has been made. Positive smears, while of invaluable aid in arriving at a diagnosis of Vincent's infection and other types of ulcerative gingivitis, are by no means conclusive evidence of the disease. Clinical signs must

also be present, and briefly they are: edema of the gums, the characteristic greenish or grayish white membrane at the margins with evidence of beginning necrosis, a fetid odor, slight to intense pain, profuse bleeding on mild pressure, beginning destruction of the interdental papillae, looseness of the teeth, occasionally slight temperature rises and slight leucocytosis, malaise, and nervousness. After the acute stage has been passed, certain instrumentation is essential and this is as important a factor in producing a cure as any part of the treatment. Following application of local medication, we have advised that zinc peroxide be used by the patient at home every 3 hours for the first 24 to 48 hours, then every 4 hours. It is recommended that the zinc compound be mixed with water to form a creamy paste. This can be done in an ordinary medicine glass. One-half teaspoonful for each application is sufficient. This paste is applied by the patient with his finger or on a sterile swab, if desired, and massaged gently into the interproximal spaces between the teeth and along the gingival margin. We think that the finger does a better job and advise the use of a rubber stall to prevent spreading the disease. The zinc peroxide thus is placed directly in contact with the tissues and remains there for a long period of time, giving off its oxygen. No effort should be made to rinse the mouth and, as the powder is slowly mixed with the saliva, the patient should expectorate this residue. We have advised all patients not to swallow the compound, although there has never been any evidence of systemic disturbances from swallowing the small quantities which remain after the bulk has been expectorated. The fact that zinc chloride might be formed in the stomach was pointed out to Dr. W. F. vonOettingen, director of the Haskell Laboratories of Industrial Toxicology, in Wilmington, Del., and it was his opinion that it would be very difficult to introduce enough to produce untoward effects. Patients are seen every other day, when possible, for further local treatment; the zinc peroxide is continued until complete healing has taken place, and then for another week as an added precaution.

We have reported two cases of necrosis following radiation in the treatment of malignancies. In both of these cases we placed the zinc peroxide paste directly into the pockets which had been formed, allowing it to be washed away by the saliva but irrigating the wound thoroughly with saline solution before each new application. We have always used water as a vehicle to make the paste. Pennoyer[10] suggests the use of hydrogen peroxide instead of water, such as he did with good results in a case of ulceration involving the neck near the angle of the jaw.

The cases studied by us may be classified as follows:

Acute Vincent's infection	10
Chronic Vincent's infection	8
Acute gingivitis	12
Suppurating periodontoclasia	3
Radiation necrosis	2
	35

Five cases are chosen from those reported above, one from each class:

CASE 1.—Mrs. R., aged 27 years, an Italian, presented at the dispensary of the Wilmington General Hospital on Tuesday for extraction of three teeth in the maxilla. She wore a full upper denture and the three teeth were found beneath this appliance. The areas around these teeth were thoroughly infected; gingivae over the entire mouth were inflamed, with beginning necrosis about the lower gum margins and some sloughing. Pus could be elicited on pressure from the tissue about the upper teeth. Smears were positive for Vincent's organisms and a large number of short-chain streptococci were present. The breath was fetid; there was marked malaise, with temperature normal. Patient was highly nervous and cried with pain when the infected tissues about the teeth of the upper jaw were touched. The gums were thoroughly swabbed with chromic acid, 8 per cent, and zinc peroxide given for home use. For the first twenty-four hours a mouthwash consisting of 1 teaspoonful of zinc peroxide to ¼ glass of warm water was prescribed to be used every hour. This was supplemented by the paste, once daily before going to bed. After the first day, the same wash was prescribed every three hours and the paste once daily. The patient returned to the clinic on Friday and appeared much better; considerable of the inflammation having disappeared, the foul odor was gone, and her mental attitude was decidedly better. Chromic acid was again applied and the zinc peroxide continued. The following Tuesday, when seen again, the infected teeth were removed without hesitation under nitrous oxide-oxygen anesthesia, all symptoms of the marked infection having disappeared. There were still evidences of slight inflammation about the lower teeth, where the gums still had a fine, red line at the gingival border. All the necrotic tissue had been exfoliated, and the remaining tissues had taken on a normal pink color. Five teeth were removed, three in the maxilla and two in the mandible. The patient was seen the following week and had had no postoperative disturbances. The teeth required scaling and this was advised, there being no facilities at the hospital for this service.

CASE 2.—Mr. C. B., aged 35 years, came to the office on Saturday morning complaining of sore gums. Inspection showed them to be markedly inflamed; a fetid odor was present, and there was extensive marginal necrosis about all of his lower teeth. He wore an upper removable bridge and a small lower one of the same type. There was a small fixed bridge in the anterior area of the lower jaw, replacing the two central incisors and the right lateral incisor. The remaining upper teeth all showed evidence of inflammation about their necks. He obviously had a very severe Vincent's infection and reported that he had been treated for the disease some time previously. Smears were positive for Vincent's infection. There was profuse bleeding from a half-dozen places on touching the gums with cotton applicators. A definite border of grayish white necrotic tissue was noted in the posterior buccal area of the mandible on the left side and this was quite deep. The patient had noticed bleeding first about two weeks before. He had no generalized pain but complained of slight tenderness in the affected posterior area. Temperature was normal. Treatment was begun with chromic acid, 8 per cent, and ultraviolet light applied all over the mouth for ½ minute to each area. This was followed by instructions for home care with zinc peroxide, 1 teaspoonful to ¼ glass of warm water every 3 hours. Zinc peroxide paste was substituted after the first 48 hours and used every three hours. The following Monday, he was much improved. The odor had disappeared and so had the tenderness. The light was applied for intervals of 1 minute to each area and acriflavine sponged carefully into the interproximal spaces and around the gums. The patient worked in another city and could not be seen again until the following Friday, when his mouth was nearly well. The necrotic

areas had sloughed out and granulations had started. There was no odor and no bleeding; tissues were pink and all signs of inflammation had disappeared. Superficial scaling was done at this time and acriflavine and the light applied as previously. He continued to use the paste four times a day for three weeks, when final scaling and polishing were completed. This case was not seen again for six months, when, upon periodical check, the mouth tissues were found to be quite healthy and he required only routine prophylaxis.

CASE 3.—Miss T. G., aged 27 years, came to the office for routine prophylaxis. Examination revealed extensive loss of tissue about the necks of all of the teeth with marked pocket formation between the bicuspids and molars. Patient complained of bleeding upon brushing these teeth. Where inflammation had occurred in this area, granulations had formed, breaking the interdental papillae. Thorough scaling of the teeth and curettage of the granulations were accomplished and this was accompanied by marked hemorrhage. The tissues were treated locally with tincture of metaphen (does not stain) and ultraviolet light. The ulatrophia was present in the anterior part of the mouth, but the gums were quite healthy, being continuous from labial to lingual with no break in their continuity. The papillae were lost, of course. Zinc peroxide was ordered for home use as a paste, every 4 hours. At the end of two weeks, during which time the patient was not seen, examination showed no evidence of gingival inflammation except in the very depths of the posterior pockets. The gum had healed continuously from buccal to lingual throughout the mouth. The deep pockets were then cauterized with the actual cautery (platinum wire loop) and the zinc peroxide continued for another week, when the patient was discharged. There was no evidence of bleeding on brushing and the tissue color was light pink, firm to touch.

CASE 4.—Mr. D. B., aged 28 years, came to the office with a marked case of periodontoclasia. Pus could be demonstrated on slight pressure all around the teeth. His breath was offensive. Radiographs showed extensive loss of bone about all of the upper teeth and around several of the lower molars. Treatment was started with chromic acid and ultraviolet light in conjunction with sodium perborate. After three weeks there was slight improvement, but it was still possible to express pus from around the teeth and a slight odor was still present. On changing to zinc peroxide, applied every 3 hours as a paste, there was a noticeable improvement at once, the evidence of pus rapidly disappearing and the odor leaving immediately. An attempt was made to eliminate the pocket formation about the teeth on the upper right side, at this time, surgically, and the tissue healed well, exposing considerable of the roots of the teeth. After three weeks this area had completely healed, but the patient decided not to attempt any further treatment of the upper teeth, having been convinced by somebody that extraction of those particular teeth was the only permanent cure. We believe a good many of them could have been retained to form support for a partial denture which would have given him service for a long time. His lower teeth are decidedly better. There is no evidence of pus about them and the gums are healthy. Two teeth were extracted in the lower jaw. The only place we could observe any evidence of pus at the time he decided to forego further treatment was about the upper left lateral incisor and that tooth has lost nearly all of its bony support. The upper teeth have not as yet been extracted!

CASE 5.—Mr. W. S., aged 53 years, was seen with Dr. Hynes in the Carpenter Memorial Clinic, Wilmington, Del. He had been operated on for a squamous cell carcinoma along the lower left alveolar ridge and this had been followed with deep x-ray therapy. Considerable necrosis of the buccal mucous membrane followed the treatment and an area about 4 cm. by 2 cm. along the external surface of the mandible continuous with the cheek had sloughed out to a depth of 1 cm. The area

rapidly became putrescent and all effort to check the necrosis had failed. The odor given off from this region was extremely foul. Smears showed Vincent's organisms and a mixed group of staphylococci, streptococci, and bacilli. No cultures were made. The patient had become highly irritable and required large doses of codeine daily for relief from the excruciating pain which seemed to accompany the invasion of his cheek. We painted the area thoroughly with acriviolet and then applied zinc peroxide in a creamy paste, all over the pocket, every half-hour. This area was irrigated with saline solution before each new application, washing away all débris. In three days we were able to pick off large pieces of necrotic tissue and as these were removed we could see fine, pink granulations beginning within the depths of the wound. The fetid odor disappeared at once. The very large area involved became smaller and approximated the exposed portion of the mandible in ten days. It has remained that way. An external opening occurred spontaneously and radiographs showed beginning osteomyelitis. A series of pictures demonstrates the progress of this infection and we have just discovered a pathologic fracture in the area of the mental foramen. A definite line can be seen around the involved bone extending along the border of the mandible to the angle of the jaw and thence upward along the line of the ascending ramus. This piece should sequestrate shortly. During all of the time (six months) since the original zinc peroxide treatment was employed, there has been no recurrence of soft tissue necrosis and no further soft tissue has been lost. The wound is kept clean by occasional washings with saline solution. It is our policy not to attempt to remove the involved bone until it actually becomes quite loose and we are awaiting that turn of events. At the end of one month after the zinc peroxide treatment was instituted, the patient required no sedative.

CONCLUSIONS

1. Zinc peroxide (ZnO_2) has been used clinically in 35 cases of mouth infection.

2. Its oxygen content and pH have been compared with hydrogen peroxide and sodium perborate.

3. Its ability to remain in contact with mouth tissues and to continue to give off oxygen over a long period of time have been demonstrated to be of considerable value in destroying anaerobic bacteria and some other organisms associated with diseases of the oral mucous membrane and periodontium.

4. It is nonirritating to the oral mucosa when used either as a creamy paste or diluted to the consistency of a mouthwash.

5. It is a powerful deodorant.

6. It produced no harmful systemic effects, although its chemical composition immediately suggests that patients should be advised not to swallow it.

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SEYMOUR HADEN, SURGEON AND PAINTER-ETCHER

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IT IS safe to say that there have been thousands of doctors who, though they followed after Aesculapius, have looked with covetous eyes upon the nine Muses. To be sure most pill-bearers and lusty wielders of the scalpel have remained technically true to their chosen profession, but there have been literally scores who have turned aside to woo one or the other of the beautiful goddesses, for the ranks of the medical profession have always been crowded with would-be dilettantes. Candidly, it must be admitted the would-be lovers have been generally received with cold and haughty shoulders and only a relatively few have even been partially successful in their amours. Literature has proved the most susceptible. There have been many, many men who have written and written well. Oliver Wendell Holmes with his *Chambered Nautilus* was one, Sir Arthur Conan Doyle, the creator of Sherlock Holmes, another. In modern times, Warwick Deeping, Somerset Maugham, and A. J. Cronin are three of the English school whose pens and typewriters have been rated far greater than their percussion hammers or hypodermic syringes.

Other medicos have tried their luck at original musical composition, though with less success. With the exception of Borodin, the Russian, it is hard to name a doctor who was a top-flight musician and composer and, while there were and are many surgeons who have painted and etched well enough to be splendid amateurs, there has been only one man, Seymour Haden, who reached the peak in this, his adopted field.

The founder of the Royal Society of Painter-Etchers and Engravers was born Sept. 16, 1818, the son of Charles and Emma Haden. The elder Haden was a physician who, after his public school education at Rugby, left his native city of Derby and came to London. The Rugbian appears to have been alert and receptive to new ideas and is best remembered because he quickly realized the value of the epoch-making discovery of Laennec and helped to introduce the stethoscope into England. By popularizing this invention the Englishman did much to advance British medicine. The etcher's mother came from a family of musicians and was herself of better-than-average talent. Born into the higher class of English society, at that time so smug and exclusive, the younger Haden had all of the benefits of formal education (as well as its vices) that money and position could give.

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After a general schooling at Derby, and Christ Hospital, at the age of 19, the young man attended the University College of London. The next year the youth furthered his professional education at the Sorbonne. This was followed by additional studies in Grenoble where a minor position, that of prosecteur, was secured on the faculty of an army medical institution. To cap off this peripatetic education, the youthful student annexed another degree in Paris in 1840.

It is related of Haden that at Grenoble he endeavored to change the dull, drab routine of dissection by altering the usual supine positions of the cadavers that were used by his class. Some the young instructor had hung by the heels, others were placed in a kneeling position, and

Fig. 1.—Self-portrait by Seymour Haden. (Courtesy of the New York Metropolitan Museum.)

so on. With the bodies in such grotesque attitudes, with a gesture more Gaelic than British, he would then whip a rapier through a cadaver and immediately ask his luckless students what structures had been pierced with the corpse in this position and how such a puncture wound could and would be different with the body in another place with regard to the shift of the various muscles, fascias, and organs!

It was in France that the English physician first appeared to have started taking formal art lessons in the Sorbonne government school of art. He had drawn as a child, of course, but without instruction. In defending this digression, the recent medical graduate declared that he did so only because he was impressed with the value of good drawing as a valuable adjunct to medicine and surgery! In '43 the former

prosecteur journeyed to Italy with two friends, one of whom was a painter of better than average ability. Sketches were made by all en route.

With his influence in Rome there was no difficulty in securing repeated audiences with the current Pope and much to his Catholic friends' horror the surgeon surreptitiously succeeded in sketching His Holiness' likeness on the cuff of his shirt. Gregory XVI is said to have drily remarked upon being informed of this break of etiquette that he now understood why Mr. Haden had attended three successive audiences without a change of linen!

Luck and chance are mighty factors in this world, in spite of what statisticians, moralists, and adherents of predestination would have us believe. Certainly in one summer hour in Milan they changed Haden's career—or at least made it very much easier. In that city one day the young traveler was just on the point of entering his hotel when he was accosted by a greatly excited and perturbed Englishman who asked if he knew of the whereabouts of a doctor in the vicinity. Haden replied that he, himself, was a surgeon and proffered his services somewhat diffidently, remarking as he did so that an older, more experienced man was probably wanted. But no, it seemed that the two sons of a British peer had been stricken with typhoid. One had just died, the other was moribund. Help was needed immediately. The young graduate agreed to attend the sick youth. Their carriage rolled furiously out of the city to a neighboring country villa. On its arrival news was received of the death of the second boy. The parents were heartbroken. Haden could do nothing except to give a little advice as to how the bodies could be properly shipped back for burial in England. However, the father and mother, Lord and Lady Hay, took a great liking for the young doctor. So grateful and pleased were they that they promised that should he commence practice in the fashionable West End of London, they would refer all their friends to him as patients. This they did and the young surgeon prospered exceedingly. At twenty-four Haden qualified as a member of the Royal College of Surgeons, and for thirteen years deserted his etching needles and plates while he built up and held a very lucrative practice.

As the Britisher grew older his fame spread and he was called here and there by the rich. Once, indeed, the consultant had special trains, carriages, and gondolas on a professional call from London to Venice to take care of a sick fellow-countryman who had fallen ill in the latter city. With success came a manner which his enemies labeled (to use only their mild adjectives) as conceited and egotistical. It certainly must be admitted that the surgeon etcher had no shrinking-violet personality.

In 1847 Haden married Deborah Delano Whistler, a daughter of Major Whistler, U. S. A., and a half-sister of James McNeill Whistler.

Her nickname was Dascha. Mrs. Haden was almost immediately thrown into the unwished-for role of peacemaker for her brother and husband, both magnificent superegotists, quarreled, at first intermittently and then continually, with each other. At first these fights were smoothed over by the surgeon's wife, but a particularly violent outburst in 1867 separated the two men for good.

What positive medical contributions did Haden have to his credit? At least two things. One was the founding and establishment of the Royal Hospital for Incurables. Another was the championship of ovariotomy, a procedure introduced to the medical world by the Kentuckian, Ephriam McDowell, and perfected by Lawson Tait, a contemporary of Haden. At one time he was vice-president of the London Obstetrical Society. At another period we note that the busy doctor was in charge of the medical and surgical instruments shown at a British Exhibition. We find him considered well enough professionally to be the private physician of William Makepeace Thackeray. Indeed, Haden attended the distinguished author in his final illness.* As far as definite advances go, the Englishman's research work is, frankly, for modern eyes, of questionable value. One finds records of a paper on cholera and another published in the British *Lancet* in which he recommends and advocates the use of leeches for the condition of amenorrhea. These were to be applied to the os uteri via speculum and to be followed by a hip bath. Conjecture arises upon reading the reports whether or not these cases were actual pregnancies or whether the enthusiastic bloodletter had not unwittingly produced abortions on his patients with the subsequent return of the normal flow! But the investigation for which the famous etcher is best recalled is really unique for such a supposed lover and creator of the beautiful and exquisite. We find him publicly arguing for "a proper disposal of the dead" and his brochure "Earth to Earth" is a plea for a more "rational observance of the condition essential to proper burial. . . ." The paper strenuously opposes advocates of cremation who "would drive" the "vapour of the bodies of 3,000 people who died weekly in London alone, at a needless cost, an infinite waste, and on such a scale with ill results out into the respirable air of the city." And as for human ashes: "How foolish to put in urns or scatter broadcast over our fields." "No, much better," argues our etcher-surgeon, "let us have papier-maché coffins and place these where the bodies can immediately return to the earth." In proof of this, the essayist writes that on Christmas Day, 1870, he buried his favorite dog without benefit of any casket, vault, or similar paraphernalia. When exhumed in November of 1874, nothing was found of the deceased animal

*He knew Dickens intimately, met Browning and Tennyson socially, and had a nodding acquaintance with Carlyle. (One wonders if the dour Scot ever asked his advice about his famous duodenal ulcer. It is probable that he did not for the essayist appears to have enjoyed his ill health too greatly.)

but a few bones. In addition to this bit of experimental work Haden seems to have carried out other (what were to him at any rate) conclusive arguments and experiments that to a jaundiced mind certainly savor a trifle of necrophilia! But at any rate, the surgeon believed enough of his views to debate openly in the journals of the day against cremationists and against those who favored vaults and brick graves. He attacked with ponderous ridicule those individuals who argued that these last mentioned methods should always be used in order to give those persons who might still be alive when buried a chance to be saved, should they come to after they had been pronounced dead!

And just what did Seymour Haden contribute to the fine arts? Two things: First was a popularization and revival of etching. It was largely through his efforts that the Royal Society of Painter-Etchers and Engravers was founded. The personnel of the Royal Academy (that mutual admiration society founded by Joshua Reynolds) with many others, however, refused to recognize this group and applauded Ruskin when he called etching "a blundering art." Haden was later blackballed when he was proposed for membership to the older association. This was an action that greatly worried the physician, for by this time he had become accustomed to laudation and admiration from his own set and not accustomed to setbacks.

Second, there are Haden's own creative efforts which are distinguished by a masterful simplicity in craftsmanship. Many of his works rank favorably with the best of Rembrandt or anyone else who attempted this medium. This is not the time or the place for a detailed discussion of etching as a master craft, but in brief it may be said that Haden always sought to produce his ideas on copperplate by a few suggestive lines. This was in direct contrast to the "line engravers" who emphasized complete detail of the variety not unlike that produced by a photographer. For that reason he was severely criticized by many. In a book entitled *About Etching,* the surgeon collected a list of what he considered the finest etchings of all times. On glancing through this brochure the fact that Haden was a doctor and well acquainted with anatomy is immediately apparent. For example, in a description of Rembrandt's "Mother," while describing the region of the subject's eye, words and terms are used which no one but a trained anatomist would employ. Again while discussing Meryon, the ex-naval officer, etcher of moribund Parisian scenes, and a future suicide, Haden sympathetically notes that the half Englishman, half Frenchman was the son of a physician like himself. It is of interest to note that Haden did not etch any great pictures of anything related to medicine and surgery.

In common with most individuals who advocate new ideas and opinions that are contrary to prevailing ones, Haden was slow to gain recognition.

The young man first exhibited under the name of H. Dean at the Royal Academy (1860-64) without arousing any comment favorable or otherwise. It was only in a subsequent Paris exhibition that the contemporary art critic Burty spoke of the surgeon's exhibits and lauded his efforts. A bit later (1867) Haden began to print his own plates on his own press. Here the Englishman was greatly helped by the technical skill of a craftsman by the name of Goulding who was considered the peer of copperplate printers.

Curiously enough, Haden's masterpiece the "Breaking Up of the Agamemnon" was planned for a new art magazine and the profits were to be donated to the Hospital for Incurables.* Each etching immediately sold for five guineas, and the copies of the magazine are said to have grossed £4,000.

The brother-in-law of Whistler was not averse to speaking tours and came to America in 1882 to lecture on the importance of etching as a fine art. Honors began to pour in. Eighteen ninety-one saw him elected to the Athenaeum Club. Two of his sponsors were Robert Browning and Matthew Arnold. In 1894 Queen Victoria made him a knight as a reward for being the founder and first president of the Royal Society of Painter-Etchers and Engravers. Other marks of esteem included membership in the Institute of France, Société des Artistes Françoise, and the Academie des Beaux Arts. In addition to all of these the egotist managed to keep in the public eye, as has been related above, with his activities in connection with burial and cremation.

From all accounts Haden undoubtedly had a swelled head. He certainly had a right to have one, for he belonged to the small group of the world's "fortunates" in that he possessed health, luck, wealth, fame, and happiness. Born with the proverbial silver spoon in his mouth, the aristocrat's life gives the lie to the theories of our present time leaders of the New Deal and the more abundant life who declare that only the poor and threadbare can, may, and should succeed. As a medical man he was better than the average of his time. When a young man he took up his father's profession and advanced it to a certain extent in that he recognized revolutionary changes when they came. It is generally conceded that his etchings and conceptions of art succeeded in starting a renaissance in that particular field. In addition, he created masterpieces which are extraordinarily beautiful and amazingly simple and, while Haden had many failings (like everyone else), these etchings are a concrete evidence that the surgeon-etcher was as capable in actual practice as he was in talk and theory. That in itself is indeed a rare treat at any time for in this world the actual number of individuals who can honestly be so classified will always be comparatively few.

*Because of its size, it was not published with the *Portfolio* and instead the etching "Brig at Anchor" was substituted.

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Editorials

Low Back Pain and Sciatica

THE subject of low back pain and sciatica may be said to cover one of the most important phases of orthopedic surgery. The evolution of our understanding of this condition has been slow but sure. There undoubtedly are many phases of the problem which still need clarification. There is, however, unquestionably a better understanding of the condition today than there ever was in the past.

The conservative types of treatment of this condition have not been touched upon in the symposium presented in this issue. In no way should this be interpreted to mean that we or any others interested in the subject believe that it is only a surgical problem. Given ideal conditions in most cases, the patients can be relieved if not completely cured by conservative measures and others may so regulate their activities as to have relatively little trouble from the condition. We have not dealt with the many other lesions which may be the cause of backache and sciatic pain, such as inflammatory and neoplastic conditions, all of which are equally important and should be recognized.

Elsewhere in this number of SURGERY will be found articles dealing with the more important surgical approaches to this problem. The fact that the condition has been treated surgically for many years shows that in spite of many failures from such treatment there has been a strong feeling that the answer to the problem in many cases is surgical. The fact that so many persons have had trouble enough to cause them to submit to operation shows that conservative measures have failed in many cases.

The fusion operations of both the lumbosacral and sacroiliac joints have been advocated for many years, and in many cases the patients have been cured by these procedures. Failures have been frequent enough, however, to urge those interested in the subject to look for other means. Hence, the Ober fasciotomy has been demonstrated by Ober, as well as by others, to be a valuable aid in the relief of this condition. Since the work of Mixter and Barr, the rupture of the intervertebral disk, which causes pressure on the nerve roots, has been shown to be an important cause of low back pain and sciatica. Improvement in methods of the diagnosis of this condition has brought to light a considerable number of cases. Whether or not the methods

of treatment now in use are going to stand the test of time cannot now be foretold, but, at least, the lesion has been established and many more patients are now being promptly relieved by surgical treatment than were relieved formerly.

Thus, the problem, instead of being simplified, has become even more complex, and in most cases a complete neurologic investigation is necessary before decision regarding treatment can be made. There is every reason to believe, therefore, that interest in this subject is greater than it has been and that patients who are suffering from this condition are more likely to receive relief now than they ever were in the past.

—*Ralph K. Ghormley, M.D.*
Rochester, Minn.

Anti- or Prophlogistic Treatment of Acute Infections? The Role of Immobilization and Elevation in Pyogenic Infections of the Extremities

LISTER'S successes with employment of the carbolic acid dressing in the management of compound fractures led the way to a prophylactic antisepsis and asepsis. With the discovery and the growth of the new science of bacteriology, surgery broadened in its scope, extricated itself from the status of a mere handicraft to deserve to be known as a science and in turn quickened the spirit of all of medicine. Yet, Lister's innovation directed at the treatment of infected wounds made no worthwhile contribution to the problem of dealing with established infection. Infection remains still one of the major unsolved problems of medicine and surgery.

The period of the World War saw a reawakening of interest in attempts to stem asepsis by the introduction of antiseptic agents into wounds. Dakin's solution when employed with the Carrel technique promised to be an answer to the long sought remedy for infected wounds. Persons in high places pronounced eulogies over this new principle of wound management and asserted that *conquest* as well as prevention of infection had been achieved. Time and experience have proved unfortunately that these statements were not well founded. To be sure, Dakin's solution has real merit, but rather in accelerating favorable termination of a local infection after the natural defense mechanism of the body has won the conflict with the invading organisms. Dakin's solution possesses no power to overcome virulent infection or to localize it.

At the beginning of the century, with discovery of antitoxins for diphtheria and tetanus, the time seemed not far distant when a specific im-

munologic agent would be available for every bacterial disease. That promise appears now less likely of early fulfillment than it did forty years ago. Yet our ability to deal more effectually with established pyogenic infection can receive real impetus only through the elaboration of specific immunologic or pharmacologic agents. Though sulfanilamide promises to be of value in some infections, we still await the immunologic or chemical Messiah that will lead us out of the fear and bondage of suppuration.

In the meanwhile, surgeons whose daily task it is to wrestle with the problem of infection must do what they can to aid the natural defense mechanism of the body in its conflict with infection. The role of the surgeon is essentially that of a pus evacuator; when an abscess has formed, he may incise it. Incisions before localization of a suppurative process are admitted generally now to do more harm than good.

For considerably more than a hundred years, up until the last decade of the century which has passed, common practice amongst surgeons in the management of infections was to employ antiphlogistic treatment; that is, therapy was directed at reduction of the effects of inflammation. About fifty years ago, pathologists studying changes occurring in inflammation concluded that the accompanying heat and swelling were useful and helped to fix the infection locally and lessen the tendency to centripetal spread. That these deductions were largely correct has been demonstrated definitely by Menkin[5] who has observed that the local swelling incident to inflammation mechanically isolates the infection and precludes central migration of bacteria.

In the mind of Bier[2] the suggestion that the effects of inflammation served the bodily economy in a useful manner found ready acceptance and he sought to increase the heat and swelling attending infection by the application of heat (active hyperemia) and production of venous stasis (passive hyperemia). Adami[1] became also an ardent advocate of the beneficial effects of the increased temperature and swelling of inflammation. Their teachings dominated surgical practice and augmentation of heat and swelling by employment of active heat and passive hyperemia (dependency or a compression bandage) became the order of the day. This teleological concept of the purposefulness and utility of the local inflammatory effects accompanying infection, occasionally referred to as the local defense reaction, constitutes the basis of current practices in dealing with infections.

The cellular and fluid exudate accumulating in the tissue spaces in response to infection possesses undoubtedly immunological properties of great value. In addition, as Menkin[5] has shown, the swelling attending inflammation mechanically helps to keep the infection local. Yet it is not difficult to understand that tense edema accompanying infection may be harmful. The studies of Landis[3] on capillary filtration have contributed much to a better understanding of the local sequence of

events attending inflammation. Such swelling occurs first through the agency of increased capillary permeability excited by the infection. Menkin[5] has found in purulent exudates a polypeptide-like enzymatic digestion product of protein which increases capillary permeability. He has called this substance leucotaxine. The ensuant increase of tissue pressure or tension causes compression of venules with augmented venous pressure, which in turn is reflected directly by an equivalent increase in capillary pressure, an effect which augments filtration of fluid from the capillaries into the tissue spaces of the inflamed area. The factor which operates to limit such transudation is the distensibility of the tissues. When the tissue tension becomes greater than the pressure in the arterial loops of the capillaries, filtration ceases but at the expense of an inadequate oxygenation of the tissue.

Heat aggravates filtration through both normal and abnormally permeable capillaries; so does dependency, too, through the agency of increased venous pressure. To combat the edema and increased tissue tension of infection, the employment of elevation and the avoidance of heat would appear to be logical measures. Over a four-year period the writer[7] has immobilized and elevated infected extremities in plaster casts with considerable satisfaction. At first, the method was employed as a matter of last recourse where other methods had failed. During the past two years, major infections of the extremities have been treated primarily by immobilization in windowed plaster of Paris spica body casts with the extremity concerned elevated at optimal (maximal except in circulatory disturbances of the lower extremity) distance above heart level. A short incision is made only when there is definite evidence of a subcutaneous abscess. Suppurative arthritis, too, lends itself well to this mode of management. Suitable incisions through the joint capsule must be made for drainage when the plaster is applied. The patient is positioned frequently to secure good gravity drainage. Fowler's position, which has been overused greatly by surgeons, has a positive indication in carbuncular infections of the head and neck to facilitate reduction of swelling. Similarly, the Trendelenburg position is deserving of wider employment in pararectal, scrotal, pelvic, and lower abdominal infections for the same purpose. In the inflammatory edemas of long standing, the employment of effectual gravity drainage is the best means (apart from evacuation of pus by incision) of reducing swelling.

The relief of pain which attends elevation and rigid immobilization is usually striking. Muscular movement is reduced to a minimum by effectual immobilization, lessening thereby the hazard of extension of the infection through lymphatic channels. The plaster cast precludes the application of heat, apparently not a serious loss from the standpoint of the patient's comfort and a gain in the sense that neglect of the use of heat diminishes capillary transudation.

Elevation reduces the hydrostatic force of the systolic blood pressure in direct ratio to the degree of elevation of the part. The increased venous pressure consequent upon edema is diminished. Both these factors tend to reduce the capillary pressure and decrease filtration. Elevation reduces the temperature of the inflamed part also. Because of the abnormal permeability of the capillaries, edema cannot be prevented, even though elevation is instituted early. It is to be admitted also that injury of the capillary membrane by infection permits escape of plasma proteins into the tissue spaces. Elevation of an edematous extremity under such conditions, therefore, does not bring about as quick reduction of the swelling as does similar elevation of an edematous extremity in the anasarca of heart failure in which condition the protein content of the tissue fluid is usually low and the opportunity for capillary reabsorption of the filtrate much greater. Nevertheless, owing to the lessened lymph production attending immobilization and the accelerated lymph outflow from the extremity accompanying elevation, definite reduction in tissue tension as well as in volume of the extremity is observed.

Surgeons will do well to give careful consideration to the physical factors involved in the inflammatory reaction attending infection. What factors operate to bring about suppuration in an area of inflammation is a consideration to which much study must be given. It is difficult to escape the impression that apart from the necrotizing effect of bacteria, death of leucocytes terminating in pus formation is brought about in large measure by the compression effects of the swelling accompanying inflammation. Immobilization lowers the oxygen requirement of muscle and the reduction of swelling attending elevation insures a better oxygen supply to the inflamed and tense tissues. Immobilization and elevation when instituted early in infection may now and then obviate pus formation. At any rate, the suppurative process appears often to be less extensive in soft tissues when the conservative measures described here are employed and shorter incisions suffice to bring about healing. The prodding and poking of wounds with probes, hemostats, or the gloved finger are to be studiously avoided. Hudack and McMaster[4] found that the slightest scratch breaks the physical barrier of the skin, opening the lymphatic vessels, and affording opportunity for central spread of bacteria. Even gentle stroking of the skin increases the rate of diffusion of dyes from an intradermal wheal.

Whereas our medical antecedents of a century ago carried the antiphlogistic plan of treating infections a bit too far in their advocacy of the application of cold and the use of local bleeding, yet, it is not unlikely that their management had a better physiologic rationale than our prophlogistic treatment with heat and dependency. Bier's passive hyperemia is unphysiologic and should be discarded in the treatment of infections.

Clinical trial suggests that immobilization and elevation are adjunct measures of distinct value in supporting the natural defense mechanism of the body in pyogenic infections of the extremities. Their use appears to rest on a rational physiologic basis.

—*Owen H. Wangensteen.*

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Recent Advances in Surgery

Conducted by Alfred Blalock, M.D.

RECENT ADVANCES IN SYMPATHECTOMY FOR PERIPHERAL VASCULAR DISEASE

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THE discovery of vasomotor nerves was made by Claude Bernard[1] in 1851 by section of the cervical sympathetic which he found was followed by increased temperature and redness on the side of the head on which the section was made. In 1852 Bernard[2] and also Brown-Sequard[3] demonstrated that stimulation of the peripheral end of the sectioned cervical sympathetic trunk produced constriction of the blood vessels and lowered skin temperature on the side of the section. Goltz and Freusberg,[4] 1874, demonstrated in dogs that cutting nerves to the foot was followed by immediate vasodilatation and increased temperature, but in a few days the difference between the sound and operated side decreased and in a few weeks the operated side was even cooler than the normal side. This fact was not appreciated until recently in sympathetic nerve surgery for a vascular disease, although increased sensitivity of smooth muscle to adrenalin after destruction of its sympathetic nerve supply was observed by Meltzer and Auer,[5] 1904, and Elliot,[6] 1905.

DEVELOPMENT OF SYMPATHETIC NERVE SURGERY

Alexander,[7] in 1889, recommended excision of the superior cervical ganglion for epilepsy and following this many surgeons practiced this operation. Jonnesco,[7, 8] 1896, extirpated the stellate ganglion for epilepsy and for exophthalmic goiter and Jaboulay,[9] 1899, performed periarterial sympathectomy, but it was not until after the publication of Leriche[10] in 1913 that interest in this operation was aroused. Periarterial sympathectomy was based on the assumption that it interrupted vasoconstrictor fibers coursing along the arterial wall and destined for more distal parts of the arterial tree. That the sympathetic nerves to arteries of the extremities do not take such a course but run with the peripheral nerves and come off to the arteries in segmental distribution much like the corresponding sensory branches to the skin was demonstrated by Kramer and Todd[11] in the upper extremity and by Potts[12] in the lower extremity. Woollard[13] and Woollard and Phillips[14] also

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have made investigations concerning the distribution of sympathetic fibers to blood vessels and have come to these same conclusions, and it is now generally accepted that practically all the vasoconstrictor fibers of an extremity are to be found in the spinal nerves and few, if any, accompany the larger arteries for long distances towards the periphery.[15] This makes it very improbable that periarterial sympathectomy would prove satisfactory and time has tended to contribute additional evidence. Leriche's present hypothesis to explain the occasional satisfactory result is the assumption that the operation interrupts afferent nerve fibers which follow along the vessel wall.[16] There is no sound anatomic basis for this assumption.[17, 18]

Based on Boeke's researches,[19] it became more or less accepted that there was a dual innervation of skeletal muscle by the cerebrospinal and sympathetic nerves. Accepting this concept of the innervation of voluntary muscle, Royle[20] and Hunter[21] were led to publish papers in 1924 concerning the effects of sympathectomy on spastic paralysis; although, since that time, careful investigators have been unable to demonstrate a dual innervation of striated muscle[18, 22, 23] and thus establish a basis for the operation of sympathectomy in spastic paralysis, and the operation for such lesions has been abandoned. Still, these reports of Royle and Hunter proved to be the spark that set off the recent surgical attack on the sympathetic nervous system. Sympathectomy has now been used in a great variety of conditions and such a volume of clinical reports of new procedures, indications for operation, results after operation, etc., have appeared that it is necessary to limit this discussion to a small part of the subject of sympathectomy. The phase chosen for more detailed consideration is the question: Why have operations for vascular disease of the upper extremity been followed by less satisfactory results than similar operations on the lower extremity?

SYMPATHECTOMY FOR PERIPHERAL VASCULAR DISEASE

The vasodilatation, which had long before been demonstrated in animals to follow sympathetic nerve section, was not given much consideration by clinicians until similar changes in man were reported by Royle as an incidental observation following sympathectomy for spastic paralysis. Interest was aroused by this report and operations were devised and later revised for interruption of the sympathetic nerves to the upper and lower extremities in peripheral vascular disease. Disregarded were the experiments of Meltzer and Auer and Elliot which demonstrated that sensitization to adrenaline followed sympathectomy.

Through a transperitoneal approach, Adson and Brown,[24] 1925, modified and extended Royle's lumbar ramisection to include a lumbar ramisection and ganglionectomy (removal of a segment of the lumbar sympathetic trunk), and they also included a perivascular sympathetic neurectomy of the common iliac arteries. This latter part of the opera-

tion was soon discontinued.[25] Davis and Kanaval,[26] 1926, employed both the lumbar extraperitoneal approach as devised by Royle and the transperitoneal route for excision of the lumbar sympathetic trunk. In its standardized form this operation now consists in removal of a section of the lumbar sympathetic chain corresponding to the second, third, and fourth lumbar vertebral bodies.[27] Both sides may be done at one operation through the transperitoneal approach.

For denervation of the upper extremity, Davis[26] used an anterior approach similar to that employed by Royle and at first he removed all of the cervical sympathetic ganglia as well as the stellate ganglion, but later he realized that it was not necessary to remove the superior and middle cervical ganglia and then removed only the stellate ganglion. Kuntz,[28] 1927, demonstrated that from the second thoracic sympathetic ganglion there is frequently in man a communicating ramus to the lowest trunk of the brachial plexus. Adson[25] then adopted a posterior approach as described by Henry[30] for cervicothoracic sympathectomy in angina pectoris. This wider excision of the cervicothoracic portion of the sympathetic trunk was then quite generally adopted. That all the visceral efferent fibers were still not sectioned seems quite evident from the fact that Horner's syndrome sometimes did and sometimes did not follow this operation.[25] It was also noted that the results were not as good as those following lumbar sympathectomy for vascular disease of the lower extremities. The operation was then extended by Adson[31] to include more of the sympathetic trunk and ganglia in a cephalad direction. Although bilateral Horner's syndrome constantly followed this more complete operation, still the results in the upper extremity continued to be unsatisfactory according to the reports of many surgeons.[32-36] White and co-workers[35] in 1936 wrote: "Adson's reports have constituted the outstanding exception to this nearby universal feeling of dissatisfaction although even he admitted that his results in the arms were not so satisfactory as those in the lower extremities."

The following reasons have been offered to explain this difference in results following sympathectomy in the upper and lower extremities:

1. *Incomplete Sympathectomy.*—Adson,[31] 1931, wrote that suggestions of central vasomotor influence in localized cutaneous areas after his revised and more extensive posterior operation would indicate that occasionally vasomotor fibers are missed and suggested that they pass up medial to the thoracic trunk or through the cardiac plexuses to enter the cervical nerves, and he expressed the opinion that in these occasional cases it would be necessary to do a Royle type of cervical ramisectomy at a secondary operation.

Allen[37] observed that in every case of recurrence of symptoms following sympathectomy for Raynaud's disease of the upper extremity he was able to demonstrate by persistence of sweating in some part of the denervated area that some fibers were missed at operation.

Postganglionic fibers reaching the brachial plexus by a very circuitous route have been described in the cat by Kuntz.[38, 39] These fibers are described as inconstant but when present pass into the spinal canal below the second thoracic segment, ascend along the meninges and join the anterior roots of nerves forming the brachial plexus. It is suggested that these fibers, if present in man, may be a cause of incomplete sympathectomy in the upper extremity after removal of the inferior cervical ganglion and the first and second thoracic ganglia.

2. *Regeneration of Sympathetic Nerves.*—"It would appear from the work of various observers that sympathetic fibers have an uncanny power of regeneration even when barriers of fascia and muscles have been interposed."[40] But removal of the ganglia precludes the regeneration of the postganglionic fibers and the time element, ten days to three weeks,[41, 42] also makes regeneration highly improbable.

3. *Progress of Local Disease.*—This assumes that spasm in Raynaud's disease is due to a local pathologic change as suggested by Lewis.[43, 44] Here again the time element makes this highly improbable, and the close relation of recurrent spasm with emotional stimuli with almost total absence between such stimuli suggests a circulating hormone.[42]

4. *Absence of an Increased Constrictor Tonus in the Lower Extremity Sufficient to Counteract the Effects of Gravity.*—This suggestion has been made by Woollard.[45]

5. *Increased Sensitivity to Sympathomimetic Hormones.*—At the present time this seems to be the most reasonable explanation of the return of vascular spasm.

The experiments of Meltzer and Auer[5] and Elliot[6] demonstrate not only that smooth muscle becomes sensitive to adrenalin after sympathectomy but also that this sensitivity is very much increased when the ganglia are removed. When a preganglionic decentralization was performed by sectioning the cervical sympathetic trunk, the eye muscles became sensitized to adrenalin, but this sensatization was very much greater when the superior cervical ganglion was removed to produce a postganglionic denervation. This sensitization did not appear earlier than twenty-four hours after the operation, and the most sensitive preparation was obtained fourteen or fifteen days after operation. White's attention was drawn to these experiments sometime prior to 1934, and he then began to express the opinion[45, 46] that the better results in the lower extremity might be due not to a more complete sympathectomy in the lower than in the upper extremity, but rather because a preganglionic section or decentralization of the ganglia supplying fibers to the lower extremity was performed by lumbar sympathectomy, while a postganglionic denervation was done in the upper extremity by cervicothoracic sympathectomy. White and others,[35, 42, 47, 48] especially his associates, Smithwick, Freeman, Okelberry, and Whitelaw, made investigations to determine the location of the cells supplying fibers to the

upper extremity and to learn more about the nature of this sensitization of smooth muscle following sympathectomy. With Freeman, Smithwick and White[49] studied 9 patients in whom they had previously performed cervicothoracic sympathectomy. They found that by administration of epinephrine chloride of 1:250,000 dilution at the rate of 40 to 60 drops per minute, and also by the production of hypoglycemia with insulin which stimulates secretion of epinephrine, patients with a complete sympathectomy responded with spasm in the upper extremities, while patients with an incomplete sympathectomy and normal individuals did not respond with spasm. They determined that this ability of epinephrine chloride to produce spasm after complete sympathectomy appeared as early as eight days after operation but not sooner. In animals, however, after excluding the adrenals (denervation of one and removal of the other), the spasm in the denervated part (rabbit's ear) did not occur when hypoglycemia was produced.

White, Okelberry, and Whitelaw[35] made preganglionic and postganglionic section of the sympathetic nerves to rabbits' ears, and, when the rabbit was frightened or struggled or was given insulin to produce hypoglycemia, constriction appeared in the animals with postganglionic section but not in those with preganglionic section. They did similar experiments on monkeys' hands with the same results. To demonstrate that this was not due to an incomplete operation, they removed in monkeys the sympathetic chain from above the inferior cervical ganglion to below the seventh rib with results the same as those observed previously in postganglionic denervation.

OPERATIONS FOR DECENTRALIZATION OF THE SYMPATHETIC GANGLIA SUPPLYING FIBERS TO THE EXTREMITIES

Lower Extremity.—There is no absolute proof that in man the ganglionic cells for the sympathetic fibers to the lower extremity are located below the fourth lumbar ganglion, but Langley's work[50] on the cat indicates that preganglionic fibers enter the sympathetic chain through and above the second lumbar nerve and pass from the sympathetic chain as postganglionic fibers beginning with the sixth lumbar ganglion (in the cat) and then from each down to the second sacral ganglion. As they pass from the sympathetic chain, they join the roots of the sacral plexus. Also according to Langley[51] the vertebral sympathetic ganglia are segmented and each supplies sympathetic fibers to its own spinal nerve. The few fibers which do not arise from the corresponding ganglia arise from the ganglia next above or below. Hence the foot, except that very small area on the medial side supplied by the saphenous nerve, would be expected to derive its postganglionic fibers from the lowest lumbar and upper three sacral ganglia. Therefore, lumbar sympathectomy by removal of the sympathetic trunk and ganglia along the bodies of the second, third, and fourth lumbar vertebra, as this operation has been

performed since 1926, has been a decentralization of the ganglia supplying fibers to all but a very small part of the foot; that is, a preganglionic section has been the accepted operation for the lower extremity.

Upper Extremity.—White[41] found it necessary to section the anterior roots as low as the eleventh or twelfth thoracic root to completely inhibit sweating in the hand of the monkey, indicating that these fibers come from the cord low down in the trunk and synapse in the inferior cervical, the first, second, and possibly the third thoracic ganglia. This makes it improbable that the first thoracic anterior root contributes many sympathetic fibers to the upper extremity and that possibly this root does not contribute a sufficient number of preganglionic fibers to the arm to make it necessary to divide the root in decentralization of the upper extremity. On this assumption an operation was devised by Smithwick[41] and also by Telford.[40] Kuntz[39] claims to have demonstrated sweating, however, of the paw pads and constriction of the cutaneous vessels of the foot of the cat by stimulation of the first ventral root. But the clinical results obtained by Smithwick and White[46, 52, 53] would justify their assumption that this root is not important in sympathetic innervation of the upper extremity in man.

The modified dorsal sympathectomy of Smithwick[41, 52] is done through a posterior approach, removing a portion of the third rib to gain access to the posterior mediastinum where he removes a proximal section of the second and third intercostal nerves and cuts the sympathetic trunk below the third thoracic ganglion. Some of the first cases were not entirely satisfactory which was due, Smithwick believed, to an incomplete

TABLE I

RAYNAUD'S DISEASE, CERVICOTHORACIC OPERATION (REVIEW OF LITERATURE)

AUTHOR	DATE	PATIENTS	DURATION OF HEALING	RECURRENCE OF SYMPTOMS
Mayo and Adson	1932	39		5
White	1932	5		2
Allen, A. W.	1932	9		6
Flothow and Swift	1933	10	12 operations for upper extremities; 11 operations for lower extremities	7 of the 10 patients were cured
Ross	1935	20	2 yr. +	8*
White	1936	11		11
De Takats	1937	6	At least 1 winter	6
University of Minnesota Hospitals	1938	10	Average 2.9 yr.	7
PREGANGLIONIC SECTION				
Smithwick	1937	10		0
De Takats	1937	4	At least 1 winter	1

*Not considered recurrent if attacks of cyanosis occur, but are less easily induced and are not accompanied by pain.

decentralization of the upper extremity; he then extended the operation to include more of the proximal portion of the second and third thoracic nerves by carrying the dissection into the intervertebral foramen and dividing the anterior and posterior rootlets before any white rami would possibly be given off. Since this modification, the results are, according to Smithwick and White,[46, 52, 53] as good as those in the lower extremity.

Telford's operation[40] is, in principle, the same as that of Smithwick. He makes an anterior approach and cuts the white rami from the second and third nerves instead of removing a proximal segment of the nerves as done in the Smithwick operation, but Telford performs section of the sympathetic trunk below the third thoracic ganglion just as it is done in the Smithwick operation. Horner's syndrome is not produced by either the operation of Smithwick or Telford.

The results of these procedures have not been reported except by Smithwick, by White, and by De Takats,[54] but their results have been so eminently satisfactory that the procedure deserves extended trial. If the findings of Kuntz and co-workers,[39] that the first thoracic nerve contributes vasomotor and sweat fibers to the upper extremity, proves to be true in man, then it will be necessary to extend the operation of Smithwick to include section of the white ramus of this first root. It is obviously inadvisable to remove a section of this nerve on account of the important part it takes in formation of the inferior trunk of the brachial plexus. Horner's syndrome will be the inevitable result of extending the operation to include the white ramus of the first thoracic nerve. Section of the white ramus of either the first thoracic or the second thoracic

TABLE II

RAYNAUD'S DISEASE, LUMBAR SYMPATHECTOMY (REVIEW OF LITERATURE)

AUTHOR	DATE	PATIENTS	DURATION OF HEALING	RECURRENCE OF SYMPTOMS
Mayo and Adson	1932	15		0
White	1932	12 (Not all Raynaud's disease)		0
Allen	1932	4		0
Flothow and Swift	1933	10	12 operations for upper extremities; 11 operations for lower extremities	7 of the 10 patients were cured
Ross	1935	17	2 yr. +	2*
White	1936	18 (Not all Raynaud's disease)		0
De Takats	1937	2	At least 1 winter	0
University of Minnesota Hospitals	1938	3	Average 3 yr.	1

*Not considered recurrent if attacks of cyanosis occur, but are less easily induced and are not accompanied by pain.

nerve does not produce Horner's syndrome but section of rami from both the first and second nerves is invariably followed by this syndrome.[39]

RESULTS OF SYMPATHECTOMY FOR PERIPHERAL VASCULAR DISEASE

A survey of the literature for results of sympathectomy in peripheral vascular disease is disappointing in that few reports of followed cases in large numbers are to be found. Numerous reports of the physiologic changes noted soon after operation, individual case reports, and statements of opinion are found. An attempt has been made to summarize the reported cases[34, 36, 46, 52, 54-64] in Tables I, II, and III.

TABLE III

THROMBOANGIITIS OBLITERANS (REVIEW OF LITERATURE)

AUTHOR	DATE	PATIENTS	RESULTS
Ito and Asami	1931	26, lower extremities	23 good
Adson and Brown	1932	15, upper extremities 89, lower extremities	87 improved 76 useful extremity 4% amputation of extremities
Flothow and Swift	1933	4 operations of upper extremities 9 operations of lower extremities	9 of the 11 patients have been cured
Telford and Stopford	1934	6, unilateral lower extremities 42, bilateral lower extremities	25 of 48 patients good
Diez	1934	150	Recurrence in 12% of those surviving operation
Ross	1935	37	26 rest pain relieved and gangrene arrested
Leriche and Fontaine	1935	48, lower extremities	12 good more than 1 year 13 good less than 1 year
De Takats	1937	5, upper extremities (preganglionic section) 8, lower extremities	Upper, 5 excellent or good Lower: 4, excellent; 2, fair; 1, bilateral amputation; 1, postoperative death
University of Minnesota Hospitals	1938	2, upper extremities 1, lower extremities	Upper: 1, poor; 1, good 7 years after operation Lower, 1 good 4 months after operation

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Review of Recent Meetings

REPORT OF THE MEETING OF THE AMERICAN PHYSIOLOGICAL SOCIETY, BALTIMORE, MD., MARCH 30—APRIL 2, 1938

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THE annual meeting of the Federation of American Societies for Experimental Biology was held in Baltimore, Md., on March 30 and 31 and April 1 and 2, with headquarters at the Lord Baltimore Hotel. This year marks the fiftieth anniversary of the founding of the American Physiological Society, which is the parent member of the Federation, and it was announced that bound volumes of the history of the society would be distributed to each member. The president of the American Physiological Society, **Dr. Walter E. Garrey,** Nashville, Tenn., presided at the annual banquet of the Federation, which was devoted to reminiscences of the lives of H. P. Bowditch, S. Weir Mitchell, and H. N. Martin, the founders of the society.

The Federation is composed of the American Physiological Society, the American Society of Biological Chemists, the American Society of Experimental Pharmacology and Therapeutics, and the American Society for Experimental Pathology. The meetings were attended by approximately 2,000 scientists. Except for a few symposia, papers were limited to 10 minutes; 320 such papers were given in physiology, 165 in biochemistry, 47 in pharmacology, and 66 in pathology. The presentation of short papers was preceded by a joint session, at which **Dr. Nathan B. Eddy,** Ann Arbor, Mich., spoke on **The Search for More Effective Morphine-Like Alkaloids; Dr. Ernest W. Goodpasture,** Nashville, Tenn., on **Experimental Virus and Bacterial Infections of the Chick Embryo; Dr. H. C. Sherman** and **Dr. H. L. Campbell,** New York City, on **Experimental Studies on the Relation of Nutrition to Length of Life;** and **Dr. Carl J. Wiggers,** Cleveland, Ohio, on **The Dynamics of Hypertension.** Subjects discussed in symposia were anoxia, vitamin B_1, bone, and sulfanilamide.

Among features of special surgical interest was the session on tumors of the pathology society. **Dr. W. R. Franks** and **Dr. H. J. Creech,** Toronto, Ontario, showed that dibenzanthracene, an active carcinogenic compound, could be made antigenic by combining it with a protein. Mice were injected with the dibenzanthracene-protein until an immunity was produced, and such animals were more resistant than controls to the production of carcinomas by the usual procedure of painting the skin with dibenzanthracene. The ability of a normal body constituent, acetylcholine, to produce malignancies was reported by **Dr. G. E. Hall** and **Dr. W. R. Franks,** Toronto, Ontario; exhaustion of the adrenal cortex present in these animals suggested that this organ may be involved. A substance which inhibits gastric secretion was found by **Dr. Alexander Brunschwig** and **Dr. J. van Prohaska,** Chicago, Ill., in extracts of human carcinomatous stomachs; they suggest that the presence of achlorhydria in gastric carcinoma may be due to the presence of this inhibitory substance.

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The superiority of a pepsin digestion method of large quantities of diaphragm in the determination of trichinosis was reported by **Dr. L. Catron,** Ann Arbor, Mich. This method demonstrated trichina in 15 per cent of cases coming to autopsy. **Dr. S. Goldschmidt, Dr. H. M. Vars,** and **Dr. I. S. Ravdin,** Philadelphia, Pa., presented further evidence that the degree of hepatic susceptibility to chloroform is directly proportional to the amount of fat present. In general, liver damage was decreased by a high carbohydrate diet which deposited large amounts of glycogen, but, when both fat and glycogen were high, liver injury was produced as readily as when glycogen was low and fat high. **Dr. C. L. Connor** and **Dr. I. L. Chaikoff,** Berkeley, Calif., found cirrhosis in livers that were fatty as a result of pancreatectomy, and observed early cirrhotic changes when alcohol was given to animals in which fatty livers had been produced by a high fat diet.

While all phases of physiologic research were represented in the papers presented before the Federation, certain fields that are undergoing rapid development received especial attention. Predominant among these was endocrinology; recent developments in this branch have made it necessary for those interested in any aspect of metabolism to take into account the influence of the glands of internal secretion. **Dr. W. R. Campbell** and **Dr. C. H. Best,** Toronto, Canada, confirmed previous observations that the excessive administration of anterior lobe extracts can produce a prolonged diabetes mellitus. Many communications dealt with the endocrine and other factors responsible for the regulation of carbohydrate metabolism. The male and female sex hormones and the gonadotropic hormones of the pituitary also received much attention.

In the field of nerve and muscle, potential changes recorded from various areas of the brain and cord and the variations produced in such potentials by different procedures, were reported by many speakers. The vitamins also received especial attention. Nicotinic acid, which appears to be curative in pellagra, was shown to be essentially nontoxic. New fractions of vitamin D were reported, and many observations were made on the various components of the vitamin B complex.

REVIEW OF THE MEETING OF THE AMERICAN SOCIETY FOR EXPERIMENTAL PATHOLOGY, BALTIMORE, MD., MARCH 30—APRIL 2, 1938

ARILD E. HANSEN, M.D., MINNEAPOLIS, MINN.

(From the Department of Pediatrics, University of Minnesota)

V. H. MOON, of Jefferson Medical College of Philadelphia, stated that measurement of the arterial pressure fails to recognize or estimate the degree of shock, and, furthermore, shock may be well advanced and the blood pressure still be at its highest recording point. Hemoconcentration develops early and is proportional to the degree of severity of shock, judging from experimental and clinical studies. **F. M. Allen,** of the Polyclinic Medical School and Hospital, New York, N. Y., reported on experimental studies of shock in rats, produced by constriction with a tourniquet. He found that the fatal limits are defined by the amount of tissue ligated and the length of time the tourniquet is applied. Regarding the therapeutic aspects of the problem, the following observations were made: (1) early amputation following ligation saves life; (2) transfusion even from animals in shock is life saving within certain limits (thus no abundant circulating toxin is present); (3) local transudation is governed by time; (4) abundant saline solution can save

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life even when blood or acacia fails; and finally (5) most severe shock proves fatal even when the blood is kept unconcentrated with blood plasma or saline solution. His conclusions favor a toxic cause not only of shock but also of local exudation.

Several investigators were concerned with the importance of liver function under various conditions. **C. L. Connor** and **I. L. Chaikoff**, of the University of California, found that cirrhosis develops in dogs with fatty livers following pancreatectomy of long duration. The condition of the liver seemed to be identical to that of patients with cirrhosis associated with alcoholism and diabetes mellitus. **J. L. Bollman** and **F. C. Mann**, of the Mayo Foundation, found that repeated administration of carbon tetrachloride produces distortion of the hepatic architecture typical of the portal type of cirrhosis. Impaired liver function is shown by jaundice, ascites, and alteration in the serum protein. The symptoms gradually disappear, and hepatic function returns if the carbon tetrachloride is discontinued. The occurrence of an extreme degree of generalized osteoporosis in a boy 11 years of age suffering from primary carcinoma of the liver was reported by **A. E. Hansen, M. R. Ziegler**, and **I. McQuarrie**, of the University of Minnesota. There was an associated disturbance in the lipid metabolism manifest by marked lipemia involving all the fractions of the blood lipids and, at the same time, by numerous lipid-laden or xanthoma-like cells in the spleen and other organs. Mineral balance studies and blood mineral determinations, as well as repeated blood lipid studies under various conditions, such as administration of bile salts, raw pancreas, and vitamin D, together with various other observations, suggested that the liver plays an essential role in bone metabolism. **K. M. Brinkhous, E. D. Warner, W. H. Seegers**, and **H. P. Smith**, of the University of Iowa, showed that, if a fat soluble extract of alfalfa meal rich in vitamin K is fed to dogs with biliary fistula in addition to bile or bile salts, the level of plasma prothrombin (which is very low in biliary fistula dogs and is associated with the bleeding tendency) rises much more rapidly. **S. Goldschmidt, H. M. Vars**, and **I. S. Ravdin**, of the University of Pennsylvania, in working with rats, found that high carbohydrate intakes tend to minimize the injury to the liver by chloroform. The protective effect of this diet seems to be to reduce the hepatic lipid and thus to lessen the amount of chloroform absorbed. Thereby, the length of time it will exert its action on the cell protoplasm, leading to degeneration and necrosis, will be decreased. **S. C. Madden**, of the University of Rochester, studied the regeneration of blood plasma proteins by keeping the plasma protein level of the blood constant at 4 per cent by daily plasmapheresis and a constant basal diet low, but adequate, in protein. He found that, when gelatin was added to the basal diet, a slightly increased amount of plasma protein had to be removed to keep the level of the plasma protein at 4 per cent. When cystine, accompanied by either tryptophane or tyrosine or both, was added to the gelatin, greatly increased (sometimes five times) amounts of plasma protein had to be withdrawn to maintain standard conditions. Cystine alone and tryptophane and tyrosine alone or both were ineffective in increasing the plasma proteins over gelatin alone. **R. E. Knutti**, of the University of Rochester, by somewhat similar type of experiments was able to maintain dogs at low levels of total plasma protein and of fibrinogen for periods of months by repeated injections of a solution of acacia. If the injections were discontinued, the plasma proteins and fibrinogen remain low for weeks and at the same time acacia persists in the blood. It appears that dogs with Eck fistulas respond more readily to this "acacia effect" than do normal dogs. The author concludes that these findings further implicate the liver as a source of plasma protein in that the most conspicuous pathologic changes are found in the liver in these experimental animals.

Continued interest in various aspects of the cancer problem was manifest by a variety of papers presented in this general field. **M. J. Shear**, of Harvard Uni-

versity and the United States Public Health Service, presented more information concerning the carcinogenic activity of polycyclic compounds. Many different anthracene derivatives producing a variety of tumors in mice were studied. The importance of the 10-position was especially shown by crystalline 10-methyl-1, 2-benzanthracene and 10-ethyl-1, 2-benzanthracene. **W. R. Franks** and **H. J. Creech,** of the University of Toronto, studied the immunological relationship of carcinogenesis by means of the anthracene compounds. There was some evidence that a degree of cross-immunity with antigen prepared from rabbits protected somewhat carcinogenesis by methylcholanthrene in mice. These authors are now studying the effect of such antigens on spontaneous carcinogenesis. **G. E. Hall** and **W. R. Franks,** from the same institutions, reported on investigations based on the observations that large numbers of tumors developed in animals which had been given acetylcholine over prolonged periods. A variety of animals and dietary conditions have been studied, with the same effect. The adrenals showed evidence of exhaustion with beginning degenerative change. **F. H. Wiley,** of the Haskell Laboratory of Industrial Toxicology, Wilmington, Del., has continued the studies on the production of bladder tumors after the ingestion of commercial ß-naphthylamine. This type of tumor production is unique in that it develops at the side of excretion rather than the site of entrance of the causative agent. The authors, in attempting to study the compounds present in the urine, found that a large part of the material is present as organic sulfate and a small part mercopturic acid derivative. Several hundred milligrams of a compound melting at 224° C. were isolated from the urine of dogs receiving 350 mg. of ß-naphthylamine daily. The material isolated apparently was the acid sulfate of 2-amino-1-naphthol. The work of **B. Lucke** and **A. Schlumberger,** of the University of Pennsylvania, with frogs and tumor production indicated the possible existence of an organ-specific factor, probably a virus. In these studies the tumors seemed to be caused by a virus specific for the kidney. **S. Warren,** of Harvard University, found that therapeutic irradiation of the breast or esophagus for carcinoma produces changes demonstrable clinically in the lungs. Both clinical and experimental studies were made, which revealed changes in the alveolar lining cells early and latter interstitial fibrosis and thickening of the walls of the blood vessels.

Book Reviews

Surgical Anatomy of the Head and Neck. By John Finch Barnhill. Pp. 921, with 431 illustrations. Baltimore, 1937, William Wood & Co. $20.

This exhaustive treatise on applied anatomy is an authoritative contribution of real and lasting merit. To the postgraduate student of otolaryngology especially it will be of inestimable value as a practical guide to be followed closely in his anatomic dissection. The medical student, the general practitioner, and the seasoned otolaryngologist will find here a comprehensive reference work worthy of frequent perusal.

The book comprises 921 pages, of which 452 are given over to 35 chapters representing class periods of anatomic dissection. The next 460 pages contain various illustrations of anatomic studies described in the text.

The illustrations, the majority of which are the author's own drawings, depict with clarity the anatomic dissections. The numerous illustrative plates of anatomic anomalies are well chosen from the standpoint of their surgical importance and reveal the wide experience possessed by this great teacher.

The style is purposefully didactic, but it does not become wearisome. It represents the personal style of the author, lending personality and intensity to his work.

The text describes in detail a complete and orderly dissection of the head and neck especially adapted to the postgraduate student of otolaryngology. Emphasis is repeatedly placed upon the surgical importance of structures revealed during the dissection. Points of localization and possible pathways of extension along fascial planes of suppurative processes are explored with meticulous attention to the clinical and surgical significance of the structures under consideration. The outlined dissection proceeds in such a manner as to preserve important structures in their entirety, thus allowing the surgeon an opportunity to study anatomic relations in his field and to demonstrate to himself the dangers of injuring neighboring tissues during a surgical operation.

The technique of various otolaryngological operations is described and illustrated in conjunction with the anatomic study of the part.

At appropriate places discussions of normal and pathologic physiology serve to enhance the value of the book to the clinician. There is a most instructive chapter on local anesthesia in which methods of infiltration and nerve block anesthesia are described for both major and minor surgical procedures in the field of otolaryngology.

Surgical Anatomy of the Head and Neck is not only a book of practical value, but it is as well a monument to the author, Dr. John Finch Barnhill, who will long be remembered as a keen clinician and a beloved teacher.

Fractures and Dislocations for Practitioners. By Edwin O. Geckeler. Pp. 252, with 213 illustrations. Baltimore, 1937, William Wood & Company. $4.00.

This small volume has been designed to meet the needs of the recent graduate and of the practitioner who has been not too well grounded in the treatment of fractures. The author has eliminated all padding and has refrained from lengthy discussions

of controversial points, with the result that the text is a concise but reasonably comprehensive survey of modern fracture treatment. The style is simple and clear and the subject matter is well outlined. The bibliography at the end of each chapter is well chosen and suited to the purpose of the book.

The introductory chapters concerning the nature of fractures and their clinical features are sound and of considerable practical value. In discussing ways and means of immobilization, the writer has wisely devoted some space to the details of the use and handling of plaster of Paris. The more complicated methods of skeletal traction and internal fixation, together with the strictly surgical treatment of fractures, have not been included and quite properly so, as this phase obviously does not fall within the scope of this book.

The chapters dealing with the individual types of fractures of the various bones are well detailed and are remarkably complete, considering the amount of space utilized.

It would appear that the author had considered each type carefully and had chosen from the many forms of treatment available that one most generally suitable and least subject to complication and error. While exception may be taken to some of the procedures advised, it would be difficult to suggest any substitutes that would be equally foolproof. Altogether, this is a good book and should prove valuable to the student and to the man in general practice. It is to be hoped that the author, in subsequent editions, will not be tempted to enlarge the volume but will further refine and improve it within its present limits.

Chirurgische Diagnestik. By Max Kappis. Ed. 2. Pp. 736, with 545 illustrations, 5 in color. Berlin, 1937, Urban & Schwarzenberg, Publishers. Bound, 25 M.; unbound, 23 M.

After a consideration of the art of general surgical diagnosis (70 pages, Part I) the author discusses wounds, infection, ulcers and fistulae in a general way, giving an account also of the diseases of the skin, bones, joints, muscles, tendons and fascias, nerves, blood and lymph vessels (225 pages, Part II). In the final section of the book surgical diseases are considered from the regional standpoint (420 pages, Part III). The topics of the eye, ear and nose, and gynecology are omitted.

Max Kappis, the author of this monograph on surgical diagnosis, is well known to American students of surgery as the originator of paravertebral splanchnic anesthesia. This text is well done and the wide clinical experience of its author is apparent throughout the work. It is also profusely and well illustrated. This splendid work on surgical diagnosis, not so well known to American surgeons as de Quervain's *Clinical Surgical Diagnosis* (also written in German, but available in English as well), may prove a worthy rival to that text which has come to occupy so important a place on the shelves of the libraries of most surgeons. This work of Kappis' can be recommended as authoritative and up to date. It should find favor among American surgeons as well as general practitioners who desire a standard text on surgical diagnosis.